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ORIGINAL LECTURES.

REMARKS ON THE FREQUENCY AND CHARACTER OF THE PNEUMONIAS OF 1890.

An Address
delivered before the New York Academy of Medicine,
April 17, 1890.

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IN attempting to give, in the few minutes at my disposal, a statement of the prevalence and peculiarities of pneumonia as it has presented itself in Philadelphia during the epidemic of influenza, I have tried to secure some reliable statistics by addressing nearly three thousand circulars to the physicians living in Philadelphia. To these I have had 272 replies, each more or less full. Dr. Benjamin Lee, the Secretary of the State Board of Health of Pennsylvania, has kindly given me the following returns, extending up to April 5th, as made to that Board, of the pandemic of influenza:

Total number of physicians giving returns			187
" " adults affected		19,868	
" " children "		8,258	
Total			28,126
Total number of nervous (as per returns)			5,275
" " catarrhal "			13,249
" " inflammatory "			4,976
" " deaths, directly caused		36	
" " " indirectly "		147	
Total			183
Immediate cause of death, bronchitis			4
" " pneumonia			86
" " phthisis			24
" " nervous symptoms			12

Through the kindness of my correspondents, to whom I desire to return my sincere thanks for their coöperation in this inquiry, I am able to submit statistics of 35,413 cases reported as above stated by 272 practitioners. From my personal knowledge of the profession in Philadelphia, I am disposed to believe that these figures do not represent a fair average, but that I have secured returns from an undue proportion of those who treated a considerable number of cases; I believe, however, that, so universally prevalent was the disease, not less than two out of every three, or, possibly, three out of every four of the eleven hundred thousand population of Philadelphia suffered from the disease in a greater or less severity. The figures which I have secured are probably sufficiently large to warrant some deductions as to the mortality of the disease, directly or indirectly, and as to the number of cases in which pneumonia appeared as a complication. The number of deaths in the 35,413 cases is given as 257, or 0.72 per cent. Of these 257, 84 are reported as directly due to influenza without pneumonia. Out of the total number of cases of influenza, pneumonia occurred in 1485, with a mortality of 173, or 11.65 per cent. There were also reported as occurring independently of influenza, 2471 cases of pneumonia, with a mortality of 324, or 13.11 per cent. Of these, 1046 are given as croupous, with a mortality of 151, or 14.43 per cent., and 1749 as catarrhal, with a mortality of 173, or 9.89 per cent.

It will be observed that of the 28,126 cases reported to the State Board of Health by 187 physicians in various parts of Pennsylvania, there were only 86 deaths from pneumonia. No mention is made of the total number of cases of pneumonia occurring, although it is probable that under the heading "Inflammatory cases," a considerable number of pneumonias were included. The individual experience of physicians has, however, varied greatly in this respect; thus, one of the most reliable of my respondents reports 1000 cases, with only three cases of pneumonia, each case of pneumonia terminating fatally. There were 10 other fatal cases, and it may be suspected that in some of these a concealed pneumonia was the cause of death, or at least was coexistent. Another respondent, of the highest distinction as a clinical observer, reported 850 cases of influenza with but 5 pneumonias and not a single death in the entire series. It is needless to say that the experience of those largely engaged in consulting practice is wholly without value from a statistical standpoint, and the same remark must be held applicable to the returns from hospitals.

A more complete notion of the great prevalence of pneumonia during the past season may be obtained from a chart, kindly prepared for me by Dr. Frederick A. Packard from the weekly returns of the Board of Health of Philadelphia. It is seen that during the period embraced between September 7th and December 28th there were, in 1888, 428 deaths from pneumonia, and during the same period of 1889 there were 453, or about an equal number of deaths from this disease; while during the period from December 28th to March 8th the deaths from pneumonia were, in 1889, 459; and, in 1890, 810. There was also a notable, but not equal, difference in the returns of deaths from bronchitis, and it cannot be doubted that the cases of death from pneumonia in 1890 would be further increased by the fact that some such cases are included under the head of bronchitis. In addition to this, the total number of deaths returned as from influenza, without specific mention of pneumonia, is 138, and unquestionably a large proportion of pneumonias are therein concealed. Finally, it will be noticed that during the five weeks from December 28th to February 1st, the number of deaths from pneumonia were, respectively, 71, 145, 182, 131, 82, a total of 611; while during the same weeks of the previous year the figures were, respectively, 44, 50, 38, 34, 55, a total of 221. The maximum number of deaths from pneumonia in one week in the winter 1888–9 was 64—in the week ending March 8th.

In attempting to estimate the relative proportion of the croupous and catarrhal varieties, the returns made to me (and which I must regard as more reliable than the aggregate returns to the city health office) would indicate that the catarrhal were almost twice as frequent as the croupous. It is no argument against this conclusion, as would be expected, when I say that Dr. Da Costa and myself saw at least as many cases of croupous as of catarrhal, in consultation, since this might be explained by the fact that the former were the more severe and dangerous (giving a mortality of 14.43 per cent. as against 9.89 per cent.).

In passing from this merely statistical return to the peculiarities of the pneumonias of the past season, I would first note the large preponderance of cases affecting the right lung. I should be inclined to say that this side was affected at least twice as often as the left. There was also an unusual proportion of cases of apex pneumonia where the entire upper lobe was completely consolidated without implication of any other part. As usual in such cases, there seemed to be a special predominance of cerebral symptoms.

It would appear that the association with pleurisy was even more constant and marked than usual. The side pain was often intense and sudden. The pleurisies were usually plastic; in one case, coming on with pneumonia immediately after exposure during convalescence, aspiration secured a pint and a half of pus on the third day and a few ounces of sero-pus from the pericardium on the following day.

I would offer merely as a suggestion resulting from personal observation that there has been an unusual prevalence of pericarditis, both plastic and purulent, in all probability, in some cases at least, associated with and preceding influenza. Corresponding prevalence of endocarditis has not been noted.

Jaundice has been observed in Philadelphia in a considerable proportion of cases, especially in those accompanied with pneumonia of the right lung. The evidence as to the cause of jaundice is not conclusive. In the cases where careful examinations were made bile pigment was detected. In the following case (Case I.), which may be quoted in full as typical, there were hæmin crystals found in the first examination of the urine, which was made prior to the development of the jaundice, and subsequently bile pigment was detected. In the only cases where autopsies were made the bile-ducts seemed pervious, although, as is well known, a preceding catarrhal swelling may have subsided just before or after death. Jaundice was certainly a symptom of highly unfavorable omen in pneumonic cases; when it occurred merely in the catarrhal form in influenza not complicated with pneumonia it was not necessarily grave.

CASE I.—N. C., aged twenty-five, unusually vigorous and athletic, was in excellent condition when attacked by a very slight form of influenza. He was not confined to bed at all and took no medicine and did not consult a physician. He stayed in the house but went to his meals, and in four or five days announced that he felt so well that he would go out after dinner to visit a friend a few blocks distant. The night was damp and raw. After sitting and conversing about an hour he was suddenly seized with a violent pain in the right side, which grew so intense that he fell upon the floor and vomited a quantity of blackish matter. It was not said, but it is not improbable that there was blood mixed with it. The vomiting was perhaps induced by brandy and ginger which was given to him. He was taken home in a carriage and I saw him within an hour from the time of the attack, and found him with temperature over 105° and with the evidences of the congestive stage of pneumonia of the right lower lobe. He had a hypodermic of morphine to allay pain, took aconite at short intervals during the night with antipyrine and salicylate of sodium, each 5 grains. During the night pain was intense, the respirations were short and gasping and the face wore an anxious expression. The hypodermic of morphine and atropine had to be repeated. The following morning the temperature had fallen to 103° and at no time subseqently did it reach 105° till just before the fatal ending. Complete consolidation of the right lower lobe developed at the end of forty-eight hours; the cough was painful with rusty sputa; until the fourth day the temperature averaged from 102° to 103° and the pulse between 90 and 100. The stomach was irritable and retained only very small quantities of liquid. The tongue was heavily coated; the bowels were quiet. The urine was scanty, dark, and contained albumin and granular casts. He was treated with large doses of quinine by suppositories, and repeated half-grain doses of calomel until 10 grains had been taken. At the close of this time, about the fifth day, there were several loose stools of light color. The patient seemed to be doing extremely well and frequently expressed himself as improving. By this time the rapid extension of disease began and soon led to consolidation of nearly all of the upper lobe on the right side while the middle lobe remained free. On the sixth day sudden and violent pleuritic pain developed on the left side, low down, followed by rapid consolidation of the lower lobe, extending upward and producing complete consolidation of the entire lung. Deep jaundice developed on the sixth day and the urine showed the following changes:

January 12, 1890. Acid, 1031. No albumin, sugar, or casts. *Evening*—Acid, 1027. No albumin, sugar or casts. Many triple phosphate crystals.

15th. Acid, reddish color, 1024. Distinct amount of albumin. No sugar. No casts. No red blood-corpuscles. Teichmann's crystals.

16th. No hæmin crystals. Bile pigment detected by Gmelin's test.

17th. Acid. Trace of albumin. Bile pigment. No sugar. No casts. A few bile-stained leucocytes.

There were no cerebral symptoms, but a most singular persistence of a feeling of comfort, of strength and of improvement within a few hours before death; with a perfectly clear mind and a natural tone of voice he asserted that he felt well enough to sit up and walk across the room. The temperature rose progressively to 105°, the respirations steadily grew more rapid up to 50 to 60, and the pulse lost strength and grew very rapid. There was increased cyanosis. He died of heart-failure on the beginning of the eighth day. No microscopical examination of the sputa was made. The autopsy showed typical croupous consolidation of the entire left lung and, on the right side, of the entire lower lobe

and of scattered patches in the apex. The middle lobe remained entirely free but was not fully expanded. The bronchial tubes were not obstructed; on both sides there were layers of plastic lymph; there was no pericarditis; no ante-mortem clots or valvular lesions. The liver was stained yellow, its tissue presented no marked change to the naked eye and the bile-ducts seemed pervious. No lesions of the gastro-intestinal canal. The blood seemed somewhat more liquid than normal.

Albumin in the urine was, I think, found in nearly all cases of pneumonia where it was carefully looked for, though not rarely there were no casts. Both hyaline and epithelial casts were found in some cases as early as the second day. It is clear that infectious nephritis was a frequent complication of influenza and especially in cases with pneumonia.

Many cases of pneumonia were associated with an unusual degree of gastro-intestinal irritability. Vomiting frequently occurred spontaneously at the onset and was readily caused by food or medicines during the course of the disease. Diarrhœa was also often easily caused. Both of these symptoms were highly unfavorable. Hæmorrhage from the bowels occurred in one case which came under my observation where there had been marked abdominal distress and fulness. A case is quoted here in full not only on this account but as illustrating peculiar physical signs and nervous symptoms.

CASE II.—Mr. X., aged forty-six, of excellent health, but probably somewhat overtaxed and depressed in vitality, having recently suffered from a very severe carbuncle, had a sharp attack of influenza. His whole family suffered at the same time. He went out and attended to business while still relaxed and with a hard, dry cough. He exposed himself on the evening of January 8th, going out in a damp, raw air and was attacked about midnight with chills, violent pains on the right side of chest, cough, and rapid development of fever. On physical examination there was found an extraordinary weakness of respiratory murmur over the right lung, especially over the two lower lobes. It was scarcely possible to hear the vesicular murmur, and crepitant râle was developed only after cough. Even on the left side there was no compensatory exaggeration of the respiratory murmur. The case developed into typical lobar pneumonia, involving the entire posterior part of the right lung and the anterior part of its lower lobe. There were a few râles at the base of the left lung, but no pneumonia developed there. The temperature never became very high (103°, maximum). The tongue was heavily coated and the stomach was very sensitive, but he was doing apparently well when there occurred sudden collapse with marked heart-failure and abdominal distress, which was explained soon after by several bloody discharges from the bowels. There had been a great deal of hebetude and mind-wandering before this, and this increased greatly after the pain extended, so that he was desperately ill until the twelfth day, after which improvement began and increased steadily until recovery. The urine showed:

January 10, 1890. Acid, 1018. Marked amount of albumin. No sugar. No casts.

11th. Acid, 1021. Marked amount of albumin. No sugar. No casts.

13th. Acid, 1018. Marked amount of albumin. No sugar. One hyaline and one epithelial cast in three slides.

15th. Acid, 1017. Marked amount of albumin. No sugar. No casts.

The pyrexia deserved more critical study than could be given, owing to the excessive pressure of work. I would observe, in the first place, that there seemed to be a highly pyrogenic state of the system widely prevalent throughout the community during the season. I am sure that such observations as the following must be familiar to all: One of my own children, aged five, seemed somewhat dull late in the afternoon, but did not appear at all unwell otherwise. I found his temperature 104°. In the case of a child, aged twelve, whose mother is a trained clinical observer, but who merely remarked that the child seemed feverish and dull, but did not need any attention, I found the temperature 105°. The pyrexia subsided in thirty-six hours with a development of no local lesion or the evidence of a specific influenza. Naturally, therefore, hyperpyrexia would be expected in complicated cases of influenza, and very many records have been furnished supporting this view. A striking phenomenon was a very rapid rise of the temperature at the commencement of the disease; and not rarely, this initial rise continuing for the first eighteen or twenty-four hours, was followed by a distinct drop with no subsequent rise equal to the initial temperature. Temperatures of 105° and 106°, and in two cases of 107°, have been reported, the latter occurring toward the fatal close of the cases, one of whom was quite clear in his mind and the other fairly rational. On the other hand, in a few cases strikingly low temperatures were noted throughout the course of the extension of croupous pneumonia. In a case involving all of the left lung except the tip of the apex, and terminating in abscess, the initial temperature reached 102° for a brief period, but at no subsequent time was it above 101⅘° until hectic fever set in. It is proper here to call attention strongly to the fact that the vast majority of pneumonias occurred as a sequel, rather than a complication, and that they were clearly traceable to exposure to damp and raw weather while the patient had a relaxed system and while slight pyrexia persisted after the subsidence of the more marked symptoms of his influenza. Experience furnishes many instances of this fact. Both the cases above quoted are striking examples. It was not even necessary that the patient should leave the house to meet with this fate. In a marked case which I saw, a man aged thirty-five, heavily taxed with important professional work and depressed by the loss of a favorite child of three with apex pneumonia of a cerebral type, had an ordinary influenza, from which he improved so greatly by the fourth day that, in opposition to his physician's orders, he rose, dressed, and went to his library at some distance from his bed chamber. He soon had a chill, followed by temperatures of 105° and 106°, with right-sided pneumonia, rapidly developing delirium and deepening stupor, and with rapid and furious convulsions before death, which occurred on the fourth day. The practical lesson taught to all of us by this case must be embodied impressively in the records of this epidemic, and in all future cases of influenza must serve as a most important guide in the management of this disease, especially in this state of convalescence.

Allusion has already been made to the nervous and cerebral symptoms attending the influenza pneumonia of this past season. I am not aware that the cases of pneumonia which occurred unassociated with influenza presented any marked peculiarities in this respect, but, as should naturally be expected, the pneumonias which occurred during or after influenza proved otherwise. In the first place they were often associated with exceptionally severe pain about the chest and with pains in different parts of the body. There is much evidence to show that these pains might be considered partly due to general neuritis or perineuritis of varying degrees of intensity. It is not now the time to discuss the question whether this affection of nerve-trunks was of purely infectious origin or was due to atmospheric influences acting upon systems relaxed and sensitive to an almost unprecedented degree. It would seem, however, that the view of its infectious origin is strongly supported by many facts. The existence of such neural trouble has been made clear in a number of cases by muscular and sensory sequelæ. I refer to it partly in this connection because it has seemed probable to others besides myself that such a condition of the intercostal and respiratory nerves, and possibly of the pneumogastrics themselves, may be invoked to explain not only the chest pains, but the extraordinary weakness of the respiratory murmur noted in so many of our pneumonia cases. Another explanation of this striking phenomenon that has been advanced is the hypothetical enlargement of the bronchial glands. I do not know anything in direct evidence in support of this view, but merely mention as of interest in connection with the matter that Dr. John Todd, of Pottstown, Pa., reports to me that in the cases of influenza occurring in that town there was diffuse enlargement of the lymphatics, even advancing to suppuration. We had no such reports from Philadelphia.

The cerebral symptom which attended the pneumonias here were dulness, at times amounting to deep hebetude, or, in the latter stages of fatal cases, coma. In the following case (Case III.) this type of stupor was strongly marked for two days prior to the clear development of physical signs of consolidation. I am doubtful whether the case was one of influenza at all. It is more probable that it was a typical example of the ordinary infectious type of pneumonia, where, as is well known, the constitutional symptoms may precede by hours or even several days the development of the local lesions.

CASE III.—H., aged twenty-two years, an athlete in splendid condition, though overworked and exposed in a malarial district. He had felt unwell for several days but continued at business. On Friday, December 6th, he felt very tired in the evening, but put on evening clothes and went to the theatre with only a light overcoat. He had fever during the night and slept badly, but rose the following morning and went three miles to work, walking one and one-half miles. He had to go home, however, had a hard chill, and when seen that evening had a temperature of $104\frac{3}{5}°$ with evident congestion of the upper lobe of the right lung. He was stupid and heavy, but had scarcely any cough and made no complaint of pain, other than a slight soreness about the right nipple. Consolidation of the right upper lobe was not evident until December 9th. The temperature reached 105° on the second day and for five days remained between 103° and 104°. It began to fall toward the close of the seventh day and by the morning of the eighth day had reached normal. He continued in a state of deep hebetude until the sixth day, but without delirium. Throughout the whole course of the case there were only two slight coughing spells, and only on the fifth day did he raise any sputa and then but two small portions of viscid, rusty-colored exudation. A careful microscopical examination of the blood showed no plasmodium. The entire upper lobe of the right lung and part of the back of the middle lobe were solid. When the temperature fell there was rapid resolution, with complete absorption without any expectoration. There was abundant herpes at both corners of the mouth. He took 10 grains of quinine the first day and on the second day he took 20 grains in one dose in the morning. The stomach then became irritable and he was treated with carbonate of ammonium, 5 grains every three hours, and antipyrine and sodium salicylate, 4 grains each about three times daily. On the seventh day he took five doses of 5 grains of quinine each. The rapid fall in temperature which followed on the next day was probably in large part a coincidence. Quinine was continued in decreasing doses. He made a complete and rapid recovery. The urine contained albumin but no casts. Chlorides were present on every examination.

Delirium was frequently met with and varied much in type and degree. In many cases there was continual mind wandering and delirium, and such cases were not rarely followed by emotional sensibility and weakness. Another type was of wild and maniacal delirium. This came on in some cases at an early stage, even as an initial symptom; it was sometimes persistent, and in not a few cases there was subsequent temporary dementia. Lastly, in other cases, especially in neurasthenic females or in anæmic and exhausted males, there was an excited delirium with garrulousness and even with hysteroidal phenomena. In other cases there was evident irritation, meningeal or cerebral, with wild excitement, passing into stupor with convulsions; or spinal, with stiffness and retraction of the head, muscle-soreness and hyperæsthesia.

Finally, in regard to thoracic symptoms. Cough was at times almost or entirely absent (of course, generally in cases where cerebral symptoms were present). In some instances it was almost suppressed, with no adequate cerebral symptoms to explain it. Ordinarily it was a marked symptom and often severe, paroxysmal, and painful in both croupous and catarrhal forms. I regret to say that there seem to have been in Philadelphia very few careful microscopic or culture studies of the sputa; in the cases where search was made, the pneumococcus is reported to have been found. I am unable to report any new facts in regard to the pneumonic lesions. Certainly in such cases as the first, above reported, the lesions present were to all appearance the typical ones of croupous pneumonia, and the same statement is made of other cases with and without microscopic examination.

There is nothing especial about the sputum except that in cases with little or no cough even extensive consolidation terminated in rapid absorption without expectoration (as in Case III.).

The physical signs were deeply interesting and in many cases were difficult to understand. To such an extent was this the case that I doubt not that in many examinations the diagnosis as to the extent and nature of pneumonia, or even as to the existence of pneumonia at all, was erroneous. The most evident cause of this difficulty was the excessive weakness of the respiratory murmur as above mentioned, and the poor development of all auscultatory phenomena. Further, the co-existence of bronchial catarrh, even in croupous cases, often complicated the physical signs, and not only were the breath-sounds weak in the early stages of congestion, but with complete consolidation the bronchial breathing was at times imperfectly developed.

A careful observer reports that he deliberately punctured a case of croupous pneumonia to establish a differential diagnosis because the bronchial murmur was so feeble as to justify the suspicion that there might be a considerable amount of fluid. Although in many cases of the croupous type there was typical crepitus, it was often developed only on the deep breathing following coughing. In most cases, also, the râles were coarser and heard somewhat in expiration, though chiefly in inspiration, and in cases which were clearly croupous. After the exudation was absorbed, the affected area continued in some cases for a considerable time to give impaired resonance, weak breath-sounds, and fine expansion crepitus.

CASE IV.—Mrs. W. was seen in consultation with Dr. James Collins. She was a woman in good general health when she was attacked with a slight chill, followed by fever and coughing, which soon became associated with some pain, especially in the right side. There were also pains throughout the body evidently due to influenza. The fever was of the regular type, with a maximum of 104⅜° and with daily variations of from 2° to 3½°. At the close of the fourteenth day the temperature reached 100°, but did not remain down long as symptoms of increased pulmonary trouble in the form of catarrhal pneumonia developed, and for the next five days the fever ranged between 100° and 103°, dropping again on the seventeenth day to 99⅜°. The next two days presented an even greater range of remission from 104° to 99⅖°, and the following day rose from 100° to 104⅖°; from the eighteenth day to the twenty-eighth day the fever ranged from 103° to 105°, reaching 105⅛° once. It then gradually fell and reached 99° on the thirtieth day, and during convalescence did not again rise above 100°. From the thirteenth day to the thirtieth day the patient was so ill that her life was despaired of. Both lungs were extensively involved, but at no point was there any considerable area of dulness. The respiratory murmur was extremely feeble and in many places almost inaudible. At other points it was feebly blowing, with prolonged expiration. Percussion resonance on very light percussion gave very irregular results at different points of the pulmonary area. The cough was feeble and there was very little expectoration. It was not rusty-colored at any time. The pulse was extremely feeble and ranged from 96 to 136. The râles heard were irregularly distributed and at certain points, both anteriorly and posteriorly, there were quantities of small subcrepitant râles. The respirations were not very rapid in comparison with the temperature, the average being from 24 to 26 in a minute. The treatment consisted of nutritious enemata, small doses of morphine with quinine by suppositories, and by the mouth strychnine in large doses with aromatic spirits of ammonia. The patient made a complete recovery.

It is not designed to make any remarks in reference to treatment. The general type of the disease forbade the use of depletives or of depressing remedies. The general course of treatment which seems most useful may be inferred from the cases above reported.

ORIGINAL ARTICLES.

ON THE RADICAL CURE OF HERNIA, WITH RESULTS OF ONE HUNDRED AND THIRTY-FOUR OPERATIONS.[1]

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AT the meeting of last year this Association had the benefit of a thorough critical paper on the various methods of radical cure of hernia, from Dr. Mastin, who used these words in closing the discussion: "But at this date the operation which is best calculated to offer general satisfaction remains a mooted question, and until more accurate experience decides it, the selection of the special operation must still remain *sub judice*." It goes without saying, that in the solution of this problem, "accurate experience" is not the only desideratum. Observations which cover a number of years are equally important. The tendency of hernia to relapse after apparent cure was a well-established fact when mechanical treatment was the general rule of practice, and, except in the case of children, patients were encouraged after the hernia ceased to appear, to continue the use of trusses as a safeguard against relapse. In illustration of this fact Segond,[2] in his thesis on "Radical Cure," presents a table of 39 cases, collected by Berger, in which, after apparent cure by the truss, relapse was noted at periods varying from five to sixty years.

But in the commendable zeal to perfect some method of cure for this disabling conditions, operators seemed to have overlooked this tendency to relapse, and have been ready to propose one modification after another, with a confidence justified by only months of observation. Even since the experience of surgeons has shown that the majority of relapses occur within a year after radical operation,[3] there have been plenty of new methods, or justifications of older ones after a brief period of waiting.

As an illustration of this undue haste in forming conclusions, I may refer to a paper read before a

[1] A paper read at the meeting of the American Surgical Association, at Washington, May 14, 1890.

[2] Cure Radicale des Hernia, p. 356. Paris, G. Masson, 1883.

[3] Anderegg: Deutsche Zeitschrift für Chirurgie, 24, 207.

society in New York, during the past winter, in which the writer reported, as cured, some cases operated on within a year, by the "open method," approved by McBurney, with some personal modifications. In less than three months later, three of the patients were reported to the surgeon from the Hospital for Ruptured and Crippled, as having applied for trusses for the relapsed herniæ. I was, myself, an example of this short-sightedness when, in 1882, I reported to the Surgical Society of New York, 5 cures in 21 cases of reducible hernia, by Heaton's method of injection, the oldest having been observed for one year and nine months only. Within the next year all these cases relapsed.

The difficulty of tracing patients after their discharge from hospital or private treatment has undoubtedly much to do with the incompleteness of the evidence offered in support of different procedures. A patient, who at the end of a few months is known to have no relapse, is counted as cured; if he then disappears the record is completed, and he stands as a "cure," of equal significance with one which has been examined after a much longer period of observation. The number of such cures, the continuance of which varies greatly, is a prominent factor in estimating the value of any method. This is not the accurate and continued observation which is needed.

It will be of decided advantage in comparing methods in the future, if we discard altogether the word "cure," and estimate their relative merits by a statement, first, of the total number of operations with result as to recovery, wound-complications and healing, then by an enumeration of the cases which have been examined at later periods with the proportion of relapses.

It is in accordance with these considerations that I present the results of my own work during the past seven years. In many respects the data are not so complete as they ought to be; but this shortcoming seems unavoidable in work done, as this has been, in four different hospitals, with a constantly changing staff of internes. After the injection treatment (Heaton) proved so unsatisfactory, I took up in 1883, shortly after the publication of Leisrink's statistics,[1] the more modern methods. An examination of the methods on which Leisrink reported, satisfied me that the most essential feature of any effective procedure was that proposed by Riesel, viz., the ligature of the sac at its highest point. This has since been generally adopted. But there was some doubt in my mind as to the relative advantage of the closure of the pillars of the external ring with sutures, proposed by Czerny, or the plan adopted by Socin, of merely leaving the canal to granulate over the sutured integuments. In the first series of cases, 40 in number, I followed this plan. After exposure of the neck of the sac, by dividing the integuments and anterior wall of the inguinal canal, the sac was isolated and ligated at its neck with catgut; the portion below was dissected out when it was small; when large, or containing the testicle, it was merely drained through the bottom of the scrotum. The wound was sutured over a drain in the inguinal canal, which was left for three or four days. This method, which may for brevity be termed the method of "ligature and excision of the sac," has been adhered to and favorably reported on by Socin,[1] of Bâle.

In a second series of cases, 39 in number, the method has been the same with the addition of catgut sutures to the pillars of the external ring and the divided aponeurosis. This may be denominated the method of "Ligature, excision, and suture." It is Czerny's method with the addition of the high ligature of the sac, as Riesel proposed, and has been extensively employed by Banks, of Liverpool,[2] who, however, used silver wire for suture.

I compared these two methods in a paper before the New York State Medical Association, in September, 1889, and found that, estimating the "cures" and relapses in accord with the plan adopted by others, the results were almost identical. Twenty patients in each series had been followed for periods varying from six months to four years; forty per cent. had relapsed, and sixty per cent. were "cured" when heard from. All of these patients had been operated on more than one year previously, but some of them could not be found at a later period than a few months after operation. I have subjected these patients to a fresh scrutiny,[3] in the past three months, with the help of Dr. Albert H. Leyton, and, as will be seen later, more time having elapsed, the percentage of so-called "cures" has suffered no diminution.

The third series embraces 39 cases, all operated on in 1889; in these the sac had been treated as before, but the anterior wall of the canal has been more respected and only divided when it was impossible to reach the neck by drawing down the sac. But the canal has been sutured with two layers of silk or catgut sutures, the deeper ones uniting the muscular fibres, the more superfical ones the aponeurosis and the pillars of the external ring. I would call this the method of "Ligature, excision and suture of canal."

The fourth series consists of sixteen operations

[1] Die moderne radical-operation des unterleibsbruches. Hamburgh, Leopold Foss, 1883.

[1] Revue de Chirurgie, April 10, 1888, 264.

[2] British Medical Journal, December 10, 1887.

[3] In rearranging these cases, one case has been lost from the second series, making the number now 39 instead of 40, as before reported.

on children, between the ages of four and fourteen. Here no one method has been adhered to because the condition of the herniæ varied greatly. All operations have been performed within the past six months, but enough relapses have been noted within that time to warrant some conclusions.

The total number of cases may be advantageously considered with reference to indications, mortality, complications, and wound results. As to indications, of the 134 cases, 77 were reducible, 42 irreducible, and 15 strangulated. In nearly one-half, then, the radical cure was added to the operation for the relief of irreducibility or strangulation. This is in accordance with the general belief, I think, that it should be done in such cases with few exceptions. In irreducible herniæ I would except only scrotal enteroceles of long standing and voluminous contents, where the adhesions between loops of intestine are firm, and the return of the long-protruded mass is sure to be a matter of difficulty. It necessitates a prolonged operation with much bleeding and many raw surfaces, and the aperture is sure to be large and little likely to be closed long by any method of suture. In general, I believe that irreducible cases are best treated by operation; they do not bear the pressure of any truss with comfort, and are constantly liable to have fresh contents descend into the sac. It must be remembered, however, that many small irreducible ruptures, both inguinal and femoral, of a few days' duration, will undergo spontaneous reduction by rest in bed, or even while on the feet, with the pressure of a spica bandage.

In strangulated hernia, when the gut is not returned, the radical cure must naturally be omitted. When patients are too feeble to warrant any prolongation of the operation, it is undesirable.

Of the reducible cases, the majority have presented difficulties in the management of a truss, or, in a few instances, have been anxious to try the radical cure as a means of dispensing with it, or of being able to wear one with lighter pressure. There have been more of these cases in the third series, as shown by the following table:

Series.	Reducible.		Irreducible and Strangulated.
I. contains	11	and	29
II. "	21	"	18
III. "	29	"	92

All the cases in Table IV. have been reducible.

At the outset of my experience, I was disposed to operate without regard to age. The first series shows in regard to age:

2 patients under 20
6 patients from 20 to 30
10 " " 30 " 40
8 " " 40 " 50
9 " " 50 " 60
5 " over 60

Of these 40 patients, the earliest operated on, 14 were over 50. In the second series there were but 5 over 50, as I had begun to think it undesirable to attempt the radical cure at this age, unless in connection with irreducible or strangulated cases. The ages in Series II. are:

2 patients under 20.
12 patients from 20 to 30.
7 " " 30 " 40.
11 " " 40 " 50.
4 " " 50 " 60.
1 patient over 60.
2 patients not given.

In the third series of cases, the ages are about the same as in the second. The fourth series of patients were all between the ages of 4 and 14. The indication for operation was either difficulty of retaining the rupture with a truss, or absence of any improvement in its condition as the child continued under observation. In one or two instances, inability of the parents to adjust the truss, or inattention or irregular attendance constituted impediments to the mechanical treatment. In one instance, an adherent appendix, in another a thin slip of adherent omentum, in a third a testicle arrested in the canal explained the difficulty of retention.

There are three deaths in the entire number of 134 cases, two in the first series and one in the second. The third and fourth lists represent 55 cases in succession with no fatal result. The first death was (Case 5, Table I.) a man of eighty, in apparently good health, who was subjected at the same time to the radical operation for hydrocele (Volkmann's) on the opposite side. The hernia was a large, scrotal entero-epiplocele, and a portion of omentum was excised after ligating. Death resulted in twenty-four hours from the shock of slight hæmorrhage into the peritoneal cavity from an atheromatous artery in the omentum, which had been cut by the ligature. The second fatal result was in a healthy patient (Case 26, Table II.), twenty-six years of age, whom I advised to undergo the radical operation for an irreducible epiplocele; a large bunch of omentum was removed after ligating with great care, the attention of those present being called to the advantage of using heavy silk for this purpose. Bleeding occurred after the stump was replaced in the cavity; a large incision was necessary in order to withdraw the omentum and re-ligate the vessels. The ligature had slipped. The cavity was cleansed of blood and no further oozing took place, but death resulted from septic peritonitis in twenty-four hours. The third death (Case 15, Table II.) was from shock in a large, scrotal strangulated case of twenty-four hours' duration.

These fatal results are worthy of notice in so far as they serve to emphasize my conviction that old and feeble subjects should not be subjected to opera-

tion in reducible cases, and that too much attention cannot be given to management of the omentum. In cases of strangulated hernia the radical measures should be omitted, if patients are not in condition to endure safely prolongation of the operation.

The complications existing at the time of operation were a large uterine fibroid in one case, hydrocele of the tunica vaginalis in three cases. The latter condition was managed simultaneously by simple drainage with good results.

In the course of the operation there was bleeding from a faulty ligature in one case (ending fatally, as just mentioned), wound of the intestine in four instances, and division of the vas deferens in one case. The intestinal wounds were all made in separating adhesions; all were promptly sutured and no accident resulted. In one case there were five tears made in separating intestine from the wall of a suppurating sac (an irreducible case, 34, Table I.). The wounded vas deferens was not sutured; suppurative orchitis followed, with burrowing of pus in the abdominal wall. The patient was a healthy farmer, forty-seven years of age, and was eighty-five days under treatment. There were no accidents or complications among the children.

Following the operation, the general complications to be noted are acute mania in a woman sixty-eight years of age, who could not be traced after removal from hospital (Case 4, Table I.), and erysipelas in one case, which developed from a slowly healing sinus. Septic fever has been so exceptional as to be hardly worth noting, though the wounds have healed primarily in only about one-half the cases, as the following table shows:

Series.	Prim. union.	Suppur.	Died.	Not stated.
I.	16	21	2	1
II.	21	16	1	1
III.	12	14	0	13
	49	51		

IV. All wounds healed primarily but one; this was in an unruly boy of defective intellect who tore off his dressings.

By primary union is meant the occurrence of prompt healing of the wound which, with the exception of the drainage-tube opening, has closed within a few days with but a few drops of pus. Many of the cases noted as healing by suppuration were simply slow-healing drainage openings. Profuse suppuration has been rarely noted. Of the adult cases the duration of treatment on the average was 26½ days, the shortest being 10, and the longest 85 days. Bichloride irrigation and moist dressings have been uniformly employed; drains were of rubber (a few of glass); catgut was prepared according to Kocher's formula with oil of juniper.

My practice has been to make the first dressing at the end of two or three days for the purpose of removing the tubes, and to permit the second dressings to lie in place a week, if no complication occurred. Silk was used for buried sutures in about a dozen cases, boiled in five per cent. carbolic acid solution immediately before and sometimes during the operation; yet in spite of these precautions, all the cases heard from had small abscesses after leaving hospital and discharged one or more sutures.

Of the varieties of hernia, there were 116 inguinal, 15 femoral, and 3 ventral. In the different series there are reported as follows:

	Series I.	Series II.	Series III.	Series IV.
Inguinal.	26 { 9 reducible. 13 irred. 4 strang.	36 { 22 reducible. 11 irred. 3 strang.	38 { 28 reducible. 6 irred. 4 strang.	16 reducible.
Femoral.	12 { 1 red. 7 irred. 4 strang.	2 irred.		
Ventral.	2 reducible.	1 irreducible.		

The subsequent observation of these patients gives the following results as to relapse:

Of the 40 cases operated on in the first series, by the method of ligature and excision of the sac, only 22 have been traced:

	Total Number.	No Relapse.	Relapse.
Cases under obs over 5 years	1	1	0
" " " from 4 to 5 years	3	3	0
" " " " 3 " 4 "	2	1	1
" " " " 2 " 3 "	0	0	0
" " " " 1 " 2 "	9	8	1
" " " less than 1 year	7	1	6
	22	14	8

Of the 39 cases operated on by the method of ligature, excision and suture of the external ring, but 20 have been traced, with this result:

		No Relapse.	Relapse.
Under obs. from 3 to 4 years	1	0	1
" " " 2 " 3 "	4	3	1
" " " 1 " 2 "	10	8	2
" " less than 1 year	5	1	4
	20	12	8

Of the 39 cases operated on by the method of ligature, excision and suture of the canal and external ring, 20 have been followed for less than one year with the result that 11 have recurred.

Of the 16 children, all operated on within a year, 5 have already presented relapses—at the end of one month in 2 cases, two months in 1 case, three months in 1 case, and four months in 1 case. In 6 patients, the sac was cut across just below its neck, the remainder drained and the canal sutured. But 1 of these herniæ has reappeared as yet. In the 10 others, all small, the sac was not found, and the pillars of the external ring were sutured; 4 of these have relapsed.

In comparing these methods, without reference to the time of observation, we find that:

By method I., ligature and exision of sac, there are 36.36 per cent. of relapses in 22 cases.

TABLE I.—CASES OPERATED ON BY LIGATURE AND EXCISION OF THE SAC (SOCIN).

No.	Sex.	Age.	Variety and duration.	Date. Complication.	Result.	Duration of treatment.	Last observation.
1	M.	30	Inguinal.	April 8, 1883; omentum removed.	Recovered	30 days.	
2	M.	32	Strangulated inguinal, few hours. Hernia 12 years.	August 13.	Recovered	10 days; primary union.	July, 1889. No relapse. Truss.
3	F.	55	Irreducible inguinal, 3 years. Small.	March 3, 1884; omentum removed.	Recovered	53 days.	
4	F.	58	Strangulated inguinal, 2 days. Hernia 30 years.	May 25; insanity developed.	Recovered	12 days; primary union.	
5	M.	80	Double inguinal, right, 25 years; left, 3 years. Left hydrocele radical cure at same time.	July 7; omentum removed. Hæmorrhage into peritoneal cavity.	Died		
6	M.	40	Inguinal. Small.	September 11.	Recovered	15 days; primary union.	
7	M.	5	Irreducible inguinal, 4 mos. Small.	October 27; omentum and appendix removed.	Recovered	12 days; primary union.	February, 1886. Died. No relapse at time of death.
8	M.	46	Inguinal, 14 years. Herniotomy 4 years previous. Hernia returned in 2 days. Large.	November 6; omentum removed.	Recovered	18 days; primary union.	May, 1889. Relapsed three and a half years later.
9	F.	48	Irreducible femoral, 3 years. Small.	December 9; omentum removed.	Recovered	37 days.	July, 1886. No relapse. No truss.
10	F.	51	Irreducible femoral, 8 months.	December 10; omentum removed.	Recovered	42 days.	
11	M.	55	Inguinal, 8 years. Large.	January 21, 1885.	Recovered	57 days.	
12	F.	61	Strangulated femoral, 8 days. Hernia 10 years.	January 23.	Recovered	31 days.	August, 1886. Relapsed. Truss.
13	F.	38	Strangulated femoral. Hernia 3 years.	January 29.	Recovered	21 days.	July, 1886. Relapsed soon afterward. Truss.
14	M.	23	Strangulated inguinal, 12 hrs. Hernia 5 years. Radical cure 1 year ago No relapse until 12 hours previously.	February 15.	Recovered	46 days.	January, 1886. No relapse. No truss.
15	M.	35	Irreducible inguinal, 15 years.	March 9; omentum removed.	Recovered	20 days; primary union.	
16	F.	43	Irreducible femoral, 5 years.	June 29; omentum removed.	Recovered	32 days.	July, 1886. No relapse. Truss for nine months.
17	M.	18	Irreducible inguinal, life time Small.	June 30; omentum removed.	Recovered	34 days.	
18	F.	49	Irreducible inguinal, 6 years. Small. Fibroid of uterus.	July 27.	Recovered	39 days.	
19	M.	31	Irreducible inguinal, 5 years. Very large.	August 4; omentum removed.	Recovered	55 days.	January, 1890. No relapse. Truss.
20	M.	39	Irreducible inguinal, 15 years. Large. Strangulation reduced by taxis.	August 13.	Recovered	39 days.	
21	M.	28	Inguinal, 3 years.	August 25; omentum removed.	Recovered	13 days; primary union.	January, 1890. No relapse. Truss for two years.
22	F.	29	Femoral, 2 months. Small.	September 18.	Recovered	12 days; primary union	January, 1890. No relapse. No truss.
23	M.	23	Inguinal. Small.	November 17; omentum removed.	Recovered		
24	F.	61	Irreducible femoral.	January 16, 1886; omentum removed.	Recovered	Primary union.	
25	F.	60	Strangulated femoral, 3 days. Hernia 3 years.	June 16; omentum removed.	Recovered	12 days; primary union.	
26	M.	36	Irreducible inguinal, 5 years.	August 3; omentum removed. Hæmorrhage from stump in peritoneal cavity. Stump drawn out; vessel tied.	Died	.....	Autopsy. Septic peritonitis.
27	M.	71	Strangulated inguinal, 12 hrs. Hernia 12 years.	September 5; omentum removed.	Recovered	31 days.	January, 1890. No relapse. No truss.
28	M.	29	Irreducible ventral, 5 years. Small.	March 16, 1887; omentum removed.	Recovered	16 days; primary union.	
29	M.	20	Inguinal, 8 months. Small.	May 11; omentum removed.	Recovered	42 days; primary union	
30	M.	46	Ventral, 12 years. Small.	July 14; omentum removed.	Recovered	38 days.	January, 1889. No relapse.
31	M.	59	Irreducible inguinal, 20 years	September 2.	Recovered	25 days; primary union.	Relapsed in one month.
32	M.	35	Inguinal, 16 years.	May 15, 1888, omentum removed.	Recovered	51 days.	January, 1890. No relapse. No truss.
33	M.	20	Irreducible inguinal, 5 years.	May 19; omentum removed.	Recovered	24 days.	January, 1890. No relapse. Truss. Hernia on opposite side.
34	M.	47	Irreducible inguinal, 4 years. Hydrocele.	June 5; abscess of omentum and sac; intestines wounded. Drainage of abdominal cavity.	Recovered	38 days.	October, 1888. Relapsed. No truss.
35	F.	37	Strangulated femoral, 24 hrs. Hernia 6 years.	July 14; omentum removed.	Recovered	13 days; primary union.	January, 1890. No relapse. Truss.
36	M.	55	Irreducible inguinal, 4 days. Small.	July 18; omentum removed.	Recovered	85 days.	January, 1890. Relapse.
37	F.	76	Irreducible femoral, 13 years. Small.	July 26.	Recovered	18 days.	January, 1889. Relapsed. No truss.
38	M.	51	Irreducible inguinal, 35 years.	September 3; omentum and appendix removed.	Recovered	28 days.	August, 1889. Relapsed.
39	F.	60	Irreducible femoral, 4 years.	September 15; omentum removed.	Recovered	17 days; primary union.	
40	F.	49	Irreducible femoral, 6 months. Small.	December 7; omentum removed.	Recovered	11 days; primary union.	January, 1890. No relapse.

TABLE II.—CASES OPERATED ON BY LIGATURE AND EXCISION OF SAC, WITH SUTURE OF EXTERNAL RING (BANKS).

No.	Sex.	Age.	Variety and duration.	Date. Complications.	Result.	Duration of treatment.	Last observation.
1	M.	14	Inguinal. Small.	June 1, 1885; omentum removed.	Recovered	19 days; primary union.	July, 1886. Relapsed. Truss.
2	M.	45	Inguinal, 3 years. Small.	July 9.	Recovered	15 days.	
3	M.	...	Inguinal, 1 year. Small.	July 24.	Recovered		
4	F.	40	Irreducible femoral, 2 years. Small.	November 23; omentum removed, and stump sutured in ring.	Recovered	39 days.	
5	M.	36	Inguinal, 8 years.	December 28.	Recovered		
6	M.	46	Inguinal, 3 years. Small.	February 26, 1886.	Recovered	27 days; primary union.	August, 1889. Relapsed.
7	M.	63	Irreducible inguinal, 10 years.	March 11; omentum removed.	Recovered	42 days; primary union.	February, 1888. No relapse.
8	M.	43	Irreducible inguinal, 11 years.	July 31.	Recovered	36 days.	
9	M.	45	Inguinal, 34 years.	August 6.	Recovered	25 days.	
10	M.	31	Incarcerated inguinal, 3 yrs.	August 17.	Recovered	34 days.	
11	M.	23	Inguinal.	September 17; no sac found.	Recovered	36 days.	October, 1887. Relapsed.
12	M.	...	Inguinal, 25 years.	September 25; omentum removed.	Recovered	49 days; primary union.	July, 1889. Relapsed.
13	M.	...	Inguinal, 2 months. Small.	February 26, 1887.	Recovered	16 days; primary union.	
14	M.	25	Irreducible inguinal. Small.	June 11.	Recovered	21 days.	July, 1889. No relapse. No truss.
15	M.	59	Strangulated inguinal, 1 day. Hernia 2 years. Large hydrocele.	July 17; gut congested.	Died.	.....	Death from shock
16	M.	25	Strangulated inguinal, 2 days.	July 26.	Recovered	14 days; primary union.	February, 1889. Relapse 8 months later.
17	M.	50	Inguinal, 26 years.	August 1.	Recovered	25 days; primary union.	
18	M.	22	Strangulated inguinal, 3 days. Hernia 14 years. Small.	August 2; omentum removed.	Recovered	32 days.	January, 1889. No relapse. No truss.
19	M.	41	Irreducible inguinal, 15 years. Small.	September 5; omentum removed.	Recovered	19 days; primary union.	January, 1890. No relapse. Truss.
20	M.	22	Inguinal, 4 years Small.	October 4.	Recovered	37 days.	
21	M.	48	Inguinal, 17 years. Small.	October 5.	Recovered	11 days; primary union	December, 1888. Relapsed in six months.
22	M.	35	Inguinal, 20 years. Operation two years previously. Relapse in two days.	December 15; omentum removed.	Recovered	39 days.	July, 1888. Relapse. See Table III., No. 17.
23	M.	25	Inguinal, 19 years. Small.	January 12, 1888.	Recovered	22 days; primary union.	January, 1890. No relapse. Truss.
24	M.	43	Inguinal, 6 weeks.	January 12.	Recovered	36 days.	August, 1889. No relapse.
25	M.	28	Inguinal, 7 years.	May 3.	Recovered	27 days; primary union.	
26	M.	19	Irreducible inguinal, 18 mos.	June 19; omentum removed.	Recovered	18 days; primary union.	January, 1890. No relapse. Truss.
27	M.	26	Irreducible inguinal, 2½ yrs.	June 23; omentum removed.	Recovered	18 days; primary union.	January, 1890. No relapse. No truss.
28	M.	45	Inguinal, 10 years. Large.	June 25.	Recovered	24 days; primary union.	
29	M.	24	Irreducible inguinal, 2 years.	July 16; omentum removed	Recovered	28 days.	
30	M.	23	Irreducible inguinal, 8 years.	August 4; omentum removed.	Recovered	16 days; primary union.	January, 1890. No relapse. No truss.
31	M.	55	Irreducible ventral, 4 years. Small.	August 27; omentum removed.	Recovered	10 days; primary union.	January, 1889. Relapsed.
32	M.	38	Inguinal, 7½ years. Large.	September 1.	Recovered	41 days.	January, 1889. No relapse. Truss.
33	F.	20	Inguinal, 18 months. Small.	December 13.	Recovered	10 days; primary union.	
34	M.	36	Inguinal (left), 10 years.	December 20.	Recovered	25 days.	
35	...	...	Inguinal (right), 1 year.	December 20.	Recovered	25 days; primary union.	
36	M.	46	Inguinal (right), 6 months.	December 20.	Recovered	12 days.	January, 1890. No relapse.
37	M.	30	Irreducible inguinal, 2 years. Hydrocele.	December 24.	Recovered	15 days.	
38	M.	59	Irreducible inguinal, 39 years.	December 27.	Recovered	30 days.	
39	F.	31	Irreducible femoral, 8 years.	January 2, 1889.	Recovered	15 days; primary union.	January, 1890. No relapse. No truss.

By method II., ligature and excision of sac, and suture of external ring, there are 40 per cent. of relapses in 20 cases.

This is a very trifling difference, and seems to my mind to prove that there is little choice between the two procedures. The first method has been the longer on trial by two years. It is simpler and more rapidly executed.

But the results of the first two methods are about the same, when the period of observation is taken into account. Of the cases under observation for less than a year, six out of seven relapsed in the first,

TABLE III.—CASES OPERATED ON BY LIGATURE, EXCISION, AND SUTURE OF CANAL (SILK OR CATGUT).

No.	Sex.	Age.	Variety and duration.	Date. Complication.	Result.	Duration of treatment.	Last observation. Truss.
1	M.	21	Inguinal, 17 years.	January 3, 1889.	Recovered	35 days.	
2	M.	46	Inguinal, 1 year.	January 19.	Recovered	20 days.	
3	M.	15	Irreducible inguinal, 5 months Small hydrocele	Omentum removed.	Recovered	14 days.	
4	M.	26	Inguinal, 6 months.	February 12.	Recovered	10 days; primary union.	
5	M.	37	Inguinal, 2 years.	February 12.	Recovered	20 days; primary union.	
6	M.	35	Inguinal (right), 19 years.	February 15.	Recovered	10 days; primary union.	January, 1890. No relapse. Truss.
7	...	...	Inguinal (left), 2 years.	February 15.	Recovered	Primary union.	
8	M.	18	Inguinal (left), 15 years. Cured by truss in 7 months.	February 23.	Recovered	16 days.	
9	...	...	Inguinal (right), 3 months.	February 23.	Recovered	Primary union.	
10	M.	55	Inguinal, 18 months.	March 6.	Recovered	14 days; primary union	January, 1890. No relapse.
11	M.	25	Inguinal, 4 weeks.	March 16.	Recovered	12 days; primary union.	
12	F.	34	Irreducible inguinal, 7 years.	March 16.	Recovered	14 days; primary union.	January, 1890. Relapsed in one month.
13	M.	35	Inguinal, 5 years.	March 25.	Recovered	14 days.	
14	M.	21	Inguinal, 3 years.	April 6.	Recovered	20 days.	January, 1890. Relapsed.
15	M.	18	Inguinal, 4 years.	April 15.	Recovered	11 days; primary union.	January, 1890. Relapsed.
16	M.	35	Inguinal, 21 years.	March 9.	Recovered	11 days.	January, 1890. Relapsed. See Table II., No. 22.
17	M.	42	Inguinal, 11 years.	March 1.	Recovered	34 days.	
18	M.	18	Inguinal, 3 years.	April 11.	Recovered	25 days.	
19	M.	40	Inguinal.	April 11.	Recovered	28 days.	
20	M.	29	Strangulated inguinal.	April 22.	Recovered	57 days.	January, 1890. Relapsed.
21	M.	57	Inguinal, 8 years.	May 10.	Recovered	40 days.	January, 1890. No relapse.
22	M	37	Irreducible inguinal, 10½ yrs.	May 15.	Recovered	42 days.	January, 1890. No relapse. Truss.
23	M.	28	Strangulated inguinal. Hernia 23 years.	May 29. (Gut nicked.)	Recovered	28 days; primary union.	
24	M.	19	Inguinal, lifetime.	June 1.	Recovered	41 days.	January, 1890. No relapse. Truss.
25	M.	45	Strangulated inguinal. Hernia 4 years.	May 30. (Gut nicked twice.)	Recovered	30 days; primary union.	
26	M.	25	Inguinal, 5 years.	May 4. Wound of vas deferens; erysipelas.	Recovered	138 days; suppuration; abscess of testicle: cellulitis of abdomen.	January 1890. No relapse. No truss.
27	M.	26	Inguinal, 6 years.	August 31.	Recovered	13 days; primary union.	January, 1890. No relapse. Truss.
28	F.	17	Inguinal, 2 years.	August 31.	Recovered	21 days; primary union.	February, 1890. No relapse. Truss.
29	M.	31	Inguinal, 1½ years.	September 11.	Recovered	15 days; primary union.	January, 1890. No relapse. Truss.
30	F.	40	Incarcerated inguinal, 5 yrs.	September 11. Gut wounded four times.	Recovered	20 days; primary union.	January, 1890. Relapsed. Truss.
31	M.	35	Irreducible inguinal, 2 years.	September 9. Double hydrocele.	Recovered	29 days.	
32	M.	33	Inguinal. Operation for strangulation July 11, 1888. Relapsed Sept. 12, 1889.	September 14.	Recovered	29 days.	
33	M.	33	Inguinal, 9 years.	September 14.	Recovered	23 days; primary union.	
34	M.	19	Inguinal, 3 months.	September 18.	Recovered	13 days; primary union.	January, 1890. Relapsed in canal. Truss.
35	M.	65	Strangulated inguinal, 2 days Hernia 20 years.	September 17.	Recovered	29 days; primary union.	January, 1890. Relapsed. Truss.
36	M.	42	Irreducible inguinal, 5 days. Hernia 12 years.	October 15; omentum removed.	Recovered	82 days.	
37	M.	47	Femoral, 8 weeks.	November 9.	Recovered	20 days; primary union.	January, 1890. No relapse. Truss.
38	M.	24	Inguinal, 8 months.	November 25.	Recovered	15 days; primary union.	January, 1890. No relapse. Truss.
39	M.	21	Inguinal, 18 months.	December 28.	Recovered	10 days; primary union.	

TABLE IV.—CASES OF HERNIA IN CHILDREN OPERATED ON AT THE HOSPITAL FOR RUPTURED AND CRIPPLED 1889–90.

The duration stated by parents is of doubtful value; all the cases where sac was found were anatomically congenital, except one.

No.	Sex.	Age.	Variety and duration.	Date. Complication.	Result.	Duration of treatment.	Last observation.
1	M.	6	Congenital, inguinal, scrotal; difficult.	December 3, 1889. Suture of ring. Sac not found. Wound closed; no drain.	Recovered	Primary union.	Relapsed in scrotum at end of one month. Truss.
2	M.	8	Incomplete inguinal, 6 mos. ?	January 4, 1890. Suture of ring. Wound closed; no drain.	Recovered	Primary union.	Relapsed in 2 months.
3	M.	12	Inguinal, 5 years ?	January 7. Removal of sac. Suture of ring: scrotal drain.	Recovered	Primary union.	Relapsed in three mos.
4	M.	7	Inguinal, 2 months ?	October 30, 1889. Removal of sac. Suture of ring and canal; scrotal drain.	Recovered	Primary union.	
5	M.	12	Inguinal, 5 years ?	January 28, 1890. Suture of ring.	Recovered	Primary union.	
6	F.	13	Inguinal, small, 5 years ?	November 12, 1889. Suture of ring.	Recovered	Primary union.	March 8, 1890. Relapsed. Truss.
7	F.	12	Inguinal, 3 years ?	October 22. Suture of canal and ring.	Recovered	Primary union.	
8	M.	6	Inguinal, 6 years ? Treated twice before; wearing plaster for Pott's disease.	November 19. Excision, ligature, and suture.	Recovered	Primary union.	February 27, 1890.
9	M.	9	Congenital, inguinal.	October 30. No sac found. Suture of ring.	Recovered	Primary union.	November 25, 1890. Relapsed.
10	M.	13	Inguinal ? Small.	November 19. No sac found. Suture of ring.	Recovered	Primary union.	
11	M.	14	Inguinal ?	November 12. No sac found. Suture of ring.	Recovered	Primary union.	March 6, 1890. No relapse. Truss in daytime.
12	M.	8	Inguinal, 1 year ? Unable to hold.	May 8. Excision and suture; scrotal drain.	Recovered	Primary union.	
13	M.	4½	Inguinal, 4 years ?	February 14, 1890. Suture of ring.	Recovered		
14	M.	5½	Inguinal, 1 year ?	February 21. Suture of ring.	Recovered		
15	M.	10	Inguinal, double, 8½ years.	February 14. Left ligature and suture.	Recovered		
16	M.	13¾	Inguinal, 9 years.	February 21. Excision of sac. Suture of canal. Small hydrocele of cord incised.	Recovered	April 3, suppuration.	

and four out of five in the second. In the second year the proportion of failures is about the same, but it is much less in each. Thus of nine cases operated on by the first method only one, and of ten by the second method only two recurred. In both, the tendency to relapse during the first year is markedly shown, and the inspection of cases traced after two or three years shows a much larger proportion of cases that have not reappared.

A comparison between these two and the third method, ligature, excision and suture of the canal, can be made only roughly by noting the proportion of cases recurring during the first year. This should be compared, however, with the number of cases by the other methods that have been longer under observation. If we do this we find that:

In the first series of 22 cases, 6, or 27.27 per cent., relapsed within one year.

In the second series of 20 cases, 8, or 40 per cent., relapsed within one year.

In the third series of 19 cases, 8, or 42 per cent., relapsed within one year.

If this third method has no better showing in the first year it is not likely that it will after longer observation, and it is not, therefore, entitled to greater confidence.

Of the cases in children it may be said that they prove or disprove nothing; but the prompt recurrence in cases where the sac was not found shows the importance of obliterating it in any method of cure, as well as the uncertainty of simple closure of the pillars of the external ring with sutures.

Of the effect of the use of trusses after the radical operation I can form no positive conclusion. Of the cases which relapsed where this fact was inquired into, six used trusses, three did not. Of those which did not relapse, eighteen wore trusses, eleven used none. I have usually directed patients to wear a very light support, and shall continue to do so. It seems to me important that no strong pressure should be made upon the recently healed parts; but that, on the other hand, they should be supported against the weight of the abdominal contents.

There is no advantage shown by the comparison of these methods in the femoral and inguinal varieties of hernia. If suture of the inguinal canal or the external ring leads to no better result than that of leaving the wound to heal after a drainage-tube, we cannot hope to close the femoral-canal opening any more securely.

In eight femoral herniæ, the method of ligature and excision has given me three relapses, and five apparent cures; while in inguinal hernia there are thirteen cases, eight without and five with relapse.

Of two ventral herniæ, situated between the sternum and umbilicus in the median line, one has recurred, the other has not. Another similar hernia, treated by the same method, the aponeurosis being sutured, has recurred.

Of the ages in reference to relapse, I can merely say that the majority of relapsed cases are over forty.

In reflecting on the experience of these cases I am obliged to conclude that in these methods, which I have faithfully tried, there is no prospect of attaining a radical cure of any form of hernia, and that the majority of cases will be found to relapse, if followed for a sufficient time. I am convinced, however, that the attempt to cure is desirable, since the majority of patients are in better condition than before. This is certainly true of irreducible cases, and of all reducible ones which are subjected to operation because mechanical treatment is difficult or painful. The patient who wears his truss with comfort and safety is only bettered by the operation so long as the rupture remains out of sight. The attempt to cure is furthermore desirable, since it can be made witht rifling risk to life, except in feeble, old, or unhealthy patients, and with little chance of complications or accidents. My own results as to relapse being no better by the complicated method of suture of the ring alone, or of the ring and canal, than by the simpler method of excision of the sac after ligature, I shall confine myself to that method of operation till other procedures, which have stood the test of years, make a more promising showing.

This method has the further advantage of being applicable to all forms of herniæ.

Of the newer operations, of which there are a multitude, I have not been unmindful in framing these conclusions. Each one has its earnest advocate; with a large number of theoretical advantages, or any brief period of observation, I doubt if any, subjected to the same continued scrutiny that my methods have been, will show better results. I am becoming confirmed in that judgment day by day in witnessing the relapsed cases that apply for trusses to the Hospital for Ruptured and Crippled. Between January and October, 1889, there were forty-five such cases, and in the past nine months there have been fully as many more. I am trying to trace the history of all these patients, about 100 in number, in order to obtain accurate knowledge of the methods employed. It will be of interest to know that thus far, of the forty-five patients above mentioned, twenty-two have been thus investigated. Of these, eleven had been operated on by McBurney's method, the shortest period of relapse being one month, the longest eleven months. Two had been subjected to treatment by injection, according to Heaton, with relapse at two months and eighteen months. In four operated on by ligature and excision, recurrence occurred on the average at the end of one year. One, by Macewen's method, recurred in seven months. One relapse took place four years after ligation and excision of the sac with suture of the external ring. Another, five years after treatment, by purse-string suture and inversion of the sac. Two patients, in whom the sac was sutured at its neck, had relapses in four and five months, respectively.

These observations will, without doubt, be duplicated in the cases yet to be traced, and go to strengthen the conviction that all methods of radical cure will be found unsatisfactory.

THE ETIOLOGY OF PLEURITIS, ESPECIALLY IN ITS RELATION TO TUBERCULOSIS.[1]

BY A. A. SMITH, M.D.,

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THE frequent occurrence of pleuritis, its apparent harmlessness in some cases, its very serious character in others, and the great variety of conditions under which it may arise must make it always a subject of interest. Since the discoveries of Koch, and the interest in the whole subject of tuberculosis excited by those discoveries, the study of the etiology of pleuritis and the significance of its relation to tuberculosis have received a new stimulus. Many careful observations have been made and very different conclusions have been reached by observers.

If one follows the discussions of the past few years on the subject of the relation of pleuritis to tuberculosis, he must have been thoroughly impressed with the difficulties of arriving at a satisfactory conclusion. If, as is claimed by many excellent observers, almost all cases of pleuritis not traceable to the ordinarily recognized causes are of tubercular origin, then not only must much more care be given to the diagnosis and probable etiology, but to the prognosis and treatment. If a case of pleuritis, sooner or later, goes on to the development of pulmonary tuberculosis, it is easy to accept the view that the pleuritis was of tubercular origin. The most earnest advocates of the tubercular origin of pleuritis admit that it is very difficult to prove in any given case, unless there is evidence of tubercular lesions elsewhere.

Ehrlich is of the opinion that tubercle bacilli can be demonstrated in all exudations of tubercular nature except the sero-fibrinous.

Fränkel believes it very difficult to discover the bacillus of tuberculosis in the fluids effused into the thoracic cavity, and yet he is of the opinion that

[1] Read before the Association of American Physicians, May, 1890.

the large majority of these cases are of tubercular origin.

Von Ziemssen says that even if it is not possible to show in most sero-fibrinous pleuro-exudates the microörganisms, this is no proof of their non-existence, as they may be lost in the perforation made in the pleura by the exploring-needle, or they may only occur in the form of spores.

From October 1, 1889, to April 1, 1890, I had under special observation, with a view to careful investigation and examination of the pleural exudates and sputa, 9 cases of pleuritis in Bellevue Hospital; 5 of sero-fibrinous exudations and 4 of purulent. The fluids were repeatedly examined, and were taken from the lowest portion of the pleural sac as the situation most likely to contain the cell constituents, if any were present. In not a single instance were any tubercle bacilli found. Likewise the sputa were carefully and repeatedly examined, and in only one instance were tubercle bacilli found and that was in a case in which the exudate was purulent. This was the only evidence of tuberculosis in the case; careful physical examination failed to reveal evidences of pulmonary tuberculosis. In one case of sero-fibrinous exudation the patient was aspirated twice in October. After the second aspiration the patient's condition improved very materially until early in March, when she developed an acute febrile attack with evidences of peritonitis, and died in three weeks. The autopsy was interesting in that it showed evidences of tubercular peritonitis, a tubercular lesion in the left Fallopian tube and a few miliary tubercles on the left pleural surface of the diaphragm. No tubercular lesions were found in the lungs. It was the *right* pleural sac from which the fluid had been drawn in October. These cases are mentioned to show the difficulties in the way of determining the origin of pleural exudates. They are not given as instances of tubercular or of non-tubercular pleurisies.

The study of the results of inoculation of the pleural exudates has been unsatisfactory, at least in so far as the production of tuberculosis is concerned. The most recent observations in this direction have been made by von Ziemssen, who concludes that although fresh pleural exudates injected into the serous cavities of dogs, and into their subcutaneous cellular tissue, has not produced true tuberculosis, they have acted injuriously, "but the infecting potentiality could not be isolated."

It is well known that tuberculous sputa and tuberculous material from the lungs can be injected into the tissues of animals and produce tuberculosis. On the other hand the material from so-called tuberculous joints has only, to a very moderate extent, the power of producing tuberculosis in animals. The potentiality seems to be limited to certain tissues. A number of observers confirm these conclusions of von Ziemssen.

From a clinical standpoint some recent observations are of great interest. At a meeting of the Leeds and West Riding Medico-Chirurgical Society, January 10, 1890, Dr. Barrs read a paper on an investigation of 74 cases of pleuritis under treatment from 1880–1884 inclusive, and which had apparently recovered from pleuritis, but showing at the present time a death-rate of 57 per cent. Excluding the empyema of children, he thinks the majority of cases of pleuritic effusion are of tubercular origin. Such a series of cases lends strength to the theory of the tubercular origin of pleuritis.

Coriveaud, on the other hand, reports a series of twenty-seven cases followed for from twenty-five years to eight years, and found that none of them became tubercular after attacks of sero-fibrinous pleuritis. Such a series certainly confirms the view, held by probably the vast majority of observers, of the primary and non-tubercular origin of many cases of pleurisy with effusion.

In a paper read before the American Climatological Association, Dr. Vincent Y. Bowditch gives results of an investigation of the subsequent histories of ninety cases of pleuritis occurring in his father's practice, in thirty years, and the conclusions he arrives at, are: that a large percentage of the patients who were afflicted with pleuritis, often in apparently chronic forms, recovered their health, and have not had a recurrence of the trouble, nor developed subsequent pulmonary or other tubercular trouble.

In the last twelve years, of eleven cases of pleuritis with effusion seen in private practice, of which I have records, and the subsequent histories to date, seven were purulent and four sero-fibrinous.

Of the seven purulent, six have apparently recovered, and only one has developed tuberculosis. Five of the seven followed pneumonia. Of the four sero-fibrinous, two have apparently recovered and two developed tuberculosis. It must be admitted, however, that sufficient time has not yet elapsed to give these cases much value. The advocates of the tubercular origin of pleuritis contend that many years may elapse before the pulmonary tuberculosis manifests itself—twenty-four years is the longest period I have found reported. It is very difficult to believe that there is any connection between the pleuritis and the subsequent pulmonary tuberculosis after so long a period, but it is not difficult to find observers who can believe much more improbable associations than this. Baumgarten believes that a child may be born with tubercle bacilli in some of its tissues, and that they may remain inactive for years, or even throughout life. Verneuil goes still farther. He believes that the tubercle bacillus can

exist throughout life in a parasitic form, and then manifest itself in a succeeding generation.

It will be noticed that six of my seven cases of empyema have apparently recovered. I have made no reference to a large number of hospital and dispensary cases observed, as it is impossible to get satisfactory subsequent histories, and the cases would have no value in connection with the subject of this paper. The favorable progress of purulent pleural exudate is in accordance with the experience of von Ziemssen. He reports eighteen cases of empyema. Fifteen of these were treated, of which thirteen recovered. It should be stated that all of these cases of von Ziemssen's followed pneumonia. Many other observers have had a similar experience.

In sixteen cases of dry pleuritis more or less extensive, of which I have kept records, five have thus far developed pulmonary tuberculosis. These were cases occurring in private practice; hospital and dispensary cases are not included for the same reason that such cases were not included in my series of pleurisies with effusions namely, unsatisfactory subsequent histories. Nor were cases included in which at the time there were unmistakable evidences of pulmonary disease. The observations extend over a period of fourteen years and it may be objected that this is not a sufficiently long time to determine the question of a possible development of pulmonary tuberculosis later. Still, up to date, eleven are in such a condition as to justify the opinion that they have recovered.

During the past two years I have had observations made in 140 autopsies in which there were evidences of old or recent changes in the pleura, all cases being included in which such changes were found. These autopsies were performed on subjects from Bellevue and Charity Hospitals, the hospitals of the workhouse and almshouse of the city of New York, and a few were cases of sudden death (coroner's cases) occurring outside of hospitals. There were changes in the pleura, more or less extensive, in nearly 80 per cent. of another series of autopsies, numbering 120, in which this was looked for, not including those cases where only a few fibrous threads connected the surfaces of the pleura. These changes consisted for the most part either in old adhesions, tubercular pleuritis, or recent acute pleuritis secondary to some other disease. These 120 cases were taken at random from autopsies made by Dr. Hermann M. Biggs at different hospitals in New York.

In the 140 cases 47, or 33.5 per cent., died from pulmonary tuberculosis, or else showed quite extensive pulmonary disease; of these, 9 presented unmistakable evidences of tubercular pleuritis. In 50, or a little over 35 per cent. of the remaining cases, cheesy or calcareous nodules were found in the lungs, making in all 97 cases, or 68.5 per cent. in which the lungs showed evidences of recent or old tuberculosis. Taking these two series as a whole, it is found that a little over 60 per cent. of the autopsies showed evidences of old or recent pulmonary tuberculosis, and of these less than one-half died of tuberculosis.

In 27 of the series of 140 cases the pleuritis was recent (in ten of them there were also rather extensive old adhesions), and in 5 instances it was double and associated with pericarditis. In 5 there was anthracosis with fibrous new formations in the lungs, without tuberculosis, but associated with adhesions, as a rule, rather extensive. In 16 other cases there were changes in the pleura, in which the tubercular lesions were found. Of these, 5 were cases of tertiary syphilis, 2 were associated with new growths in the lungs, 5 others showed quite marked renal disease. In one rheumatic case there was acute pleuritis, pericarditis and endocarditis; in one, chronic bronchitis and bronchiectasis; in one, an aneurism of the aortic arch, and one was a case of scleroderma with advanced heart disease.

In speaking of an old process, reference is made to the cheesy or calcareous nodules so frequently found in the lungs, and surrounded by fibrous tissue capsules. Recent investigations have clearly shown that in the vast majority of cases these nodules have resulted from a local tuberculosis; and that the cheesy masses generally, and the calcareous masses frequently, still contain the tubercular virus and will, when inoculated, produce tuberculosis in animals. It should also be distinctly understood, that the nodules found in some of the cases were very small, not more than 3 mm. in diameter, and that in some instances not more than one nodule was found in the lungs. These have been included on the ground that they are exactly the same in nature as the larger masses, and if they are tubercular the question of size does not come into consideration. Fibrous cicatrices without cheesy nodules were not taken into consideration, although many of these unquestionably result from tuberculosis.

As regards the extent and distribution of adhesions, it was found that there was no constant relation between the degree of lung involvement and the extent of the adhesions. In some cases of very advanced disease the pleura showed only a few adhesions. This was especially the case where the process did not involve to any great extent the pleural surface. On the other hand, small nodules in one lung were not infrequently associated with extensive adhesions on both sides, and often the adhesions were more marked in the lung that contained no cheesy nodules.

The observations in these cases do not confirm the assumption that the adhesions are primary, and that they predispose to the development of a

tubercular process in the lungs. The cheesy and calcareous nodules were generally found in the upper lobes, and near the apices, and were not infrequently found in other parts of the lungs also, especially in the upper portion of the lower lobes. When these nodules were found in the lungs, the bronchial glands usually showed similar changes macroscopically.

In the series of 140 cases only 9 showed unmistakable evidences of tubercular pleuritis, although 97 showed evidences of either recent or old tuberculosis in the lungs, and 47 of these showed very extensive involvement.

Post-mortem researches seem to show that tubercular pleuritis is a rare disease, even though the lungs in many cases may be extensively diseased. Undoubtedly many cases of tuberculosis have their first manifestation in a pleuritis, and many cases of pleuritis are secondary to pulmonary tuberculosis. *Possibly* a pleuritis may favor the development of pulmonary tuberculosis by depressing vitality, by lessening the proper lung expansion or by interfering with the nutrition of the adjacent lung; but more probably the same underlying cause produces both.

In the absence of evidences of tuberculosis in other situations there is no sufficiently precise clinical history to warrant an absolute diagnosis of tubercular pleuritis. If it is an effusion, the appearance of the fluid is not characteristic. Unless tubercle bacilli are found one cannot positively affirm its nature, and as has been shown the bacilli are very rarely found.

From my own researches and a study of the researches of others, I offer the following conclusions:

First. If it be true that the very large majority of cases of pleuritis, not traceable to ordinarily recognized causes, are tubercular, we have no clinical method by which the fact can be proven, and the assumption that they are tubercular can only be by inference.

Second. If such a large majority of pleurisies, as many seem to believe, are of tubercular origin, recovery from tuberculosis of the pleura is much more frequent than from pulmonary tuberculosis.

Third. The prognosis in purulent pleural exudate is more favorable as to the subsequent development of tuberculosis than in sero-fibrinous exudate.

Fourth. That even though at an autopsy, evidences of pleuritis recent or old may be found, it does not necessarily follow that the pleuritis is tubercular, because there are tubercular lesions in the lung.

Fifth. It is fair to assume from the frequent association, post-mortem, of recent and old changes in the pleura with pulmonary tuberculosis, that many of these changes are tubercular in origin, although at the time of death tubercles are not found in the pleura.

Sixth. In cases of pleuritis, although evidences of tuberculosis exist in the lungs, as shown by physical signs, and by the presence of tubercle bacilli in the sputa, it does not necessarily follow that such cases of pleuritis are tubercular in origin, although they probably are.

Seventh. In cases in which pulmonary tuberculosis develops subsequent to a pleuritis, there is probably the same underlying cause for each.[1]

ACUTE PERITONITIS FROM GONORRHŒA.

BY CHARLES B. PENROSE, M.D., PH.D.,
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PERITONITIS in women is generally the result of disease of the Fallopian tubes. The sequence of events is, in most cases, slow—salpingitis, closure of the fimbrinated end of the tube by peritoneal adhesions, and distention of the tube with pus or other material more or less septic. The resulting peritonitis, if acute and general, is caused by sudden rupture and escape of the tube-contents into the peritoneum; or, if chronic, by gradual leakage from a small rupture, or by direct extension of inflammation through the tubal or abscess wall.

In this way gonorrhœa in women often produces peritonitis and death. But the causative attack of gonorrhœa generally occurs several months or years before the final fatal peritonitis, the peritonitis being a remote sequel of the gonorrhœa, and the infecting virus being of a mixed character. The case which I am about to describe followed a very much more unusual course. The inflammatory process travelled directly from the vagina, through the previously healthy uterus, and along the Fallopian tubes, and thence throughout the general visceral and parietal peritoneum. The action was too rapid to permit the formation of preservative adhesions and occlusion of the fimbriated ends of the tubes.

Similar rapid peritonitis from a septic focus in the vagina or uterus occurs in the puerperal state, after criminal abortion, and after surgical traumatism to the uterus, as in cases reported by Coe (*N. Y. Med. Journ.*, May 15, 1886), when death followed the operation of hystero-trachelorrhaphy, and the autopsy showed septic inflammation, traceable directly from the wounded cervix, along the endometrium and tubes, and thence throughout the general peritoneum. It is, however, probable that in many cases of so-called puerperal peritonitis the rupture during labor of a previously diseased tube, or pelvic tumor of any kind, is the cause of the peritonitis, rather than septic infection from the uterus

[1] I desire to acknowledge my indebtedness for much valuable assistance in the preparation of this paper to Dr. Hermann M. Biggs, one of the Curators of Bellevue Hospital and a director of the Carnegie Laboratory.

or vagina. And it is also very probable that the same cause which was active in the case here reported—that is, gonorrhœa acquired immediately after labor—may be present, though unrecognized, in other cases of peritonitis following childbirth:

In October, 1889, a young colored man came to the Surgical Clinic of the Pennsylvania Hospital with a violent inflammatory attack of gonorrhœa, of several days' duration. He had great dysuria, a very profuse purulent discharge, inflammatory swelling of the whole penis, and œdema of the prepuce. He stated that this was his first attack of gonorrhœa, and that he had acquired the disease by being unfaithful to his wife during her convalescence from childbirth. Seven days after his first visit he had a violent attack of epididymitis on the left side, which obliged him to take to bed.

On October 29th he came at night to my office, walking with crutches, and suffering intense pain. He asked me to see his wife, who was very sick. On visiting the house I found a large colored woman, over 200 pounds in weight, presenting all the symptoms of general peritonitis. The pulse was 130. Temperature 100°. The abdomen was distended and very tender. There was a profuse purulent vaginal and urethral discharge, and redness and inflammatory swelling of the external genitals. Vaginal examination caused great pain, and revealed nothing more definite than fulness on each side of, and posterior to, the uterus. She had free diarrhœa, which had appeared a few hours after the onset of general abdominal pain, and which had greatly relieved the pain.

She and her husband gave me separately the following history. She had always been perfectly healthy, and had had two children, the last on September 24, 1889, one month before the present sickness. The child was healthy in every respect, and the mother convalesced without trouble of any kind, leaving bed on the eleventh day. She had connection with her husband, for the first time after delivery, on October 23d. This was six days before my visit to her, and, as I have said, at this time I was treating the husband for a violent attack of gonorrhœa. Coitus was followed in two days by burning micturition, swelling and inflammation of the vulva, and profuse purulent discharge. Three days later she began to have pelvic pain, greatest on the right side, and free bleeding from the vagina. This was speedily followed by general abdominal pain, tenderness, and distention. Diarrhœa appeared shortly after the general abdominal pain. The bleeding from the vagina lasted four days. As her surroundings were of the worst kind, I sent her to the Gynecean Hospital, and performed abdominal section the next day.

The parietal peritoneum was hypertrophied, being about one-eighth inch in thickness. It did not present the usual smooth surface, but was red and granular, and bled easily. The intestinal loops were adherent to the parietes and to each other in the region of the incision, throughout the pelvis, and in the right iliac and hypogastric regions. All the loops of intestine which were visible were distended, and presented the same red, granular appearance, and bled very easily, even on contact with soft sponges.

There was no lymph, pus, or fluid of any kind in the peritoneum. There were no organized adhesions, all adhesions being very recent and easily separated. The Fallopian tubes were six inches long, and as thick as the index-finger. They were bound by recent adhesions to the surrounding intestines. There was no sign of old tubal disease. The walls of the tubes were thickened, hard, and rigid. The distal ends were not closed. The fimbriæ stood out turgid and stiff. The whole organ was hypertrophied and rigid from recent inflammatory exudation throughout all the structures. A very small quantity of thin, puriform fluid was in the tubes. The tubes and ovaries were removed close to the cornua. The abdomen was freely flushed with hot distilled water, and a large glass drain was introduced. A small quantity of sanguinolent serum was discharged from the drainage-tube, which was removed on the third day.

The convalesence was uneventful. She continued to suffer from urethritis and vaginitis for five weeks after the operation, but is at present well in every respect.

The unusual course followed by the gonorrhœal disease in this case was due to two causes: The virulence of the infecting pus, which was shown by the intensity of the inflammation in the husband, and the patulous condition of the uterus and tubes incident to parturition. Both causes prevented the chronic course of the disease—the gradual formation of peritoneal adhesions and occlusion of the tubes. This would have happened if the patient had survived without operation. The operation accomplished the same result by shutting off the uterus from above, rendering the peritoneum a closed sac, safe from further infection.

I subjoin the report of the microscopic examination of the Fallopian tubes, which was made by Dr. L. Coplin, of the Jefferson Medical College Laboratory of Pathology:

"The tubes show well-marked inflammation, masked by thickening and young cell-deposit through their entire structure. Cocci are present in both tubes, the right more than the left, and in this the processes of inflammation are most developed. The cocci are not gonococci; as they are not diplococci, are not all of them within the perinuclear protoplasm, and do not stain by gonococci stain. They are larger, and are found both inter- and intra-cellular. They are rarely found singly, but as zooglœic masses, the size of which vary from $\frac{1}{2500}$ to $\frac{1}{1300}$ of an inch, and in these masses may be counted from 25 to 75 or more cocci. They have very much the appearance of some of the staphylococci of suppuration. None of them are streptococci. They are not in the epithelium of the tube, but in the younger connective tissue corpuscles of the margin of the tube. The epithelium is for the most part normal."

University of Halle.—After many erroneous announcements regarding the successor of Volkmann at Halle, the fact is that Professor Bramann is there and has begun his clinics.

CLINICAL MEMORANDA.

MEDICAL.

Case of Slow Pulse.[1]—This patient has continued in much the same condition during the past year. In November, 1889, he had a relapse of which the following is the record:

Nov. 24, 1889. Pulse 11. Frequent attacks of unconsciousness. During the attacks his pulse ceases to beat for ten, fifteen, and twenty seconds.

25th. Pulse 36. Quinine and iron prescribed.

Dec. 1. Pulse 32. Twenty attacks daily.

7th. Pulse 12. Pulse irregular for three or four beats, and then a long interval. Fainting attacks are very frequent.

13th. Pulse 34. Ammonium bromide and belladonna given.

21st. Pulse 12. Fifteen to twenty spells per day.

24th. Pulse 36. Spells not so frequent.

31st. Pulse 13. Very severe attack of syncope.

Jan. 14, 1890. Pulse 26. Very severe attack of syncope.

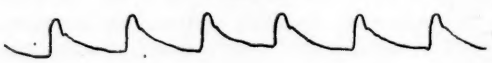
15th. Pulse 18. For a time thought to be dead.

17th. Pulse 18. Better. Temperature 99.6°.

Feb. 6. Pulse 23.

8th. Pulse 13. Slight attacks.

TRACING 1.

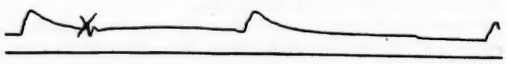


Pulse beating 36 per minute.

TRACING 2.

TRACING 3.

TRACING 4.



Tracings 2, 3 and 4 taken when pulse was beating 12 per minute They show the long diastole—5 seconds.

(Tracings were taken by Dr. R. T. Edes.)

9th. Pulse 48. At 8.20 A. M., fell on floor unconscious; black in face; no motion for one hour; choking sound in throat. At 9.15 A. M. constant rolling of the head, then jerking, kicking, and bellowing constantly as though in greatest distress. Takes no notice of surroundings, but put out tongue when told to sharply. Nitrite of amyl and hypodermic injection of morphine and atropine.

10th. Pulse 32. Recovered from attack of yesterday.

15th. Pulse 24.

21st. Pulse 28. Better and sitting up.

27th. Pulse 28. Swelling of face.

April 8. Pulse 28.

On May 14th, when the patient was brought before the Association, his pulse was 24. He was apparently comfortable and was able to be about, though he still had occasional attacks of syncope.

D. W. PRENTISS, M.D.

WASHINGTON, D. C.

[1] Patient was brought before the meeting of the Association of American Physicians, May 14, 1890. The patient was also shown to the Association in 1889. For more full account of the case, see Proceedings of The Association of American Physicians for 1889.

MEDICO-LEGAL.

Collapse of the Lung from Hanging.—Collapse of the lungs in hanging is of such rare occurrence that this case may be of some importance in legal medicine. This condition of the lungs is mentioned once in Taylor's *Medical Jurisprudence;* and I am informed by Dr. John J. Reese, Professor of Medical Jurisprudence in the University of Pennsylvania, that this is the only case recorded.

The case is as follows: B. E., colored, aged twenty-two years, was hung in this city, April 18, 1890, and was given a fall of six feet. Heart-action ceased in twelve minutes after the drop fell. Post-mortem examination held one hour afterward. Temperature in axilla at this time was 98⅘°. Body well nourished, but not very muscular.

Opening the chest, the lungs were found completely collapsed, otherwise normal.

The pleural cavities were entirely free from fluid and were without pleuritic adhesions. The right heart was distended with dark blood. The stomach contained a small amount of blood but no food, and there was marked capillary engorgement. Bowels were empty. The vessels of the liver and kidneys were engorged. Spleen normal.

Bloodvessels of brain distended.

Careful dissection of the neck showed that the articulations of the vertebræ were intact.

CUNNINGHAM WILSON, M.D.

BIRMINGHAM, ALABAMA.

MEDICAL PROGRESS.

Experimental Studies in Tuberculosis.—At the Heidelberg congress of German naturalists and physicians Schottelius stated that some years ago he instituted a series of experiments with tuberculous lungs interred in a wooden box at a depth of five feet, the usual mode of sepulture of bodies. After two years and a half he removed from the surrounding earth a quantity of tubercle bacilli, for the most part spore-producing. From these he obtained pure cultures which furnished positive results in 80 per cent. of his experiments in inoculation. He is now engaged in investigations with the object of ascertaining whether the virulence resides in the bacilli taken from the soil or in their spores. Soyka expresses the opinion that the greater number of the bacilli perish, but that

some possess durability and may recover their virulence under favorable conditions. Gärtner has observed bacilli in a cemetery abandoned for twenty-five years.

As a practical outcome of his investigations, Schottelius advises the disinfection of bodies of persons dead from infectious diseases.

Gebhardt has experimented with the sputum of consumptives, his object being to test the virulence of the sputum in different grades of dilution, and to ascertain whether the potentiality of the tubercular virus is essentially modified by the organ first infected—that is to say, by the manner of infection. With this object he practised hypodermic and intraperitoneal inoculation and experiments in inhalation and alimentation, and ascertained that in the subcutaneous connective tissue, the peritoneum, and the lungs the tuberculous virus multiplies in about an equal degree, while the digestive apparatus offers resistance. Hence tubercular virus may, especially in small quantities, pass through certain organs without provoking local alterations. As the point of ingress is not always the seat of the disease, pulmonary tuberculosis is not to be invariably attributed to infection by inhalation.

Sputum which contains bacilli is exceedingly infectious, retaining its virulence even in dilution of 1 : 100,000, apparently without regard to the manner of infection. The virulence of the sputum being in proportion to the quantity of bacilli present, Gebhardt employed pure cultures of the bacilli of tuberculosis, on the hypothesis that equal quantities of the same culture contain quantities of bacilli. With a subcutaneous inoculation of 1 c. c. of a dilution in the proportion of 1 : 400,000 and an inhalation of 0.5 c. c. of the same dilution in a culture of agar-agar he obtained positive results. Hence the pure cultures retain their virulence when enormously diluted.

Malassez and Vignal desiccated the sputum of tuberculosis, moistened it with water, again desiccated and pulverized it, and this repeatedly, endeavoring to realize, as far as possible, the conditions to which the sputum daily ejected in our streets is subjected. After successive desiccation and moistening the bacillus of the sputum retained all its virulence.

With regard to the penetration of tubercle bacilli into the organism, Dobroklonski states, as the result of his experiments in the Cornil laboratory at Paris, that tuberculosis may attack the organism by way of the digestive apparatus. For this to occur, no lesion of the intestinal wall, epithelial desquamation, local modification, nor previous inflammation is necessary. The tuberculous virus (bacilli and spores) may easily traverse the completely normal epithelial lining of the intestine, but it does not determine inflammation unless it remains for a length of time in contact with the intestinal wall. Dobroklonski asserts that the spores and bacilli do not penetrate the organism by any fixed means, but that they are carried by the current of the lymphatic system, and, being arrested by the tissues, determine in them the formation of tubercles.

As a natural inference from Hirschberger's experiments demonstrating that tuberculous cows, or cows infected with tubercular phthisis, produce, in 55 per cent. of cases, infectious milk, it was supposed that milk from large dairies would contract virulent properties from the infectious milk of one tuberculous cow. A series of experiments, conducted by Gebhardt, under the direction of Bollinger, in the Pathological Institute of Monaca, show that tuberculous milk loses its virulence at a certain dilution. The mixing of milk practised in large dairies diminishes the danger, and in most cases renders the milk innocuous. Milk served by large dairies is always to be preferred to the continued use of milk from one cow.

Studies in the transmission of tuberculosis from animals to men by means of tuberculous milk, directed the attention of hygienists to the derivatives of milk, the most important of these being butter. Gasparini inoculated guinea-pigs with butter containing the bacillus of Koch, and by microscopic observation verified tubercular lesions in almost all cases.

Referring to his studies of the infectious property in the flesh of tuberculous animals, Kastner stated that the object of his experiments was to ascertain if such flesh contained virus, and to what extent the consumption of such flesh as meat was dangerous to man. From the results of his experiments he concludes that the danger is slight, unless the nodules of tuberculosis are formed in the meat, which is rarely the case.—*Weekly Abstract of Sanitary Reports, U. S. Marine Hospital Service*, June 13, 1890.

Effects of Caffeine.—In an able article on the study of caffeine DR. EDWARD T. REICHERT reached the following conclusions:

1. The pulse-rate may be diminished during the first and last stages of the poisoning, but is generally decidedly increased. During the first stage the diminution is due to stimulation of the cardio-inhibitory centres in the medulla oblongata and heart, and during the last stage to a direct depression of the heart. The increase in the pulse-rate is due to a depression or paralysis of the above-mentioned cardio-inhibitory centres.

2. Arterial pressure during the first stages of poisoning is generally either unaffected or diminished, but occasionally a trifling increase is noted; during subsequent stages it is diminished. The increase is due to a direct stimulant action upon the bloodvessel walls increasing vascular tension. The diminution is due chiefly to a direct depression of the heart, and to some extent, doubtless, to a secondary paralysant action on the vessel walls.

3. The acceleration of the heart-beats may be accompanied by no appreciable alteration in blood-pressure, but generally by a more or less decided diminution, which is dependent upon cardiac depression.

4. Caffeine diminishes the heart's efficiency for work, arrests it in diastole, sometimes induces sudden paralysis, and is, therefore, a cardiac depressant.

5. The asserted stimulant action upon the circulation is, doubtless, subjective, and dependent upon an excitation of the cerebral centres.

The Effect of Opiates on Gastric Secretion.—According to the *Lancet*, DR. ABUTKOFF has examined the effects of opium and two of its alkaloids on the digestive functions of healthy people. He found that the three substances experimented with—viz., opium, morphine, and codeine—all exert a retarding action on gastric digestion by diminishing the general acidity of the gastric juice. Neither the quantity of pepsin nor the absorbent power of the

stomach appeared to be affected by any of the three substances. As to their relative effects in such doses as can be given medicinally, opium had decidedly the most marked influence, and codeine the least. Dr. Abutkoff suggests that in the case of patients with weak digestion, due to deficiency of acid in the gastric juice, opiates should be given some hours after a meal, but that where the hydrochloric acid is excessive, they may be given with meals.

Surgical Treatment of Tubercular Peritonitis.—CZERNY, of Heidelberg, has recently published his views upon surgical interference in cases of tubercular peritonitis. It is pointed out that though the records of operations seem to show that the disease is amenable to such treatment, it should be remembered that tubercular inflammation of a serous membrane usually indicates that tuberculosis has reached an advanced stage and has become diffused. Czerny grants that surgical treatment of tubercular lesions may, under certain circumstances, bring about an improvement in the nutritive processes, and thus aid in eliminating the virus, and, in cases of restricted localization, lead to a definite cure. It is different, however, when there are many infecting foci.

It is held that the prospects offered by surgical interference will depend upon a distinction being made between the variety of this affection characterized by the presence of a fluid exudate with miliary tubercles, and the dry form in which large tubercular nodules are found; operation in the latter variety being very unpromising. In the former variety the author regards incision and removal of the fluid much preferable to any other plan of treatment, but he also holds that the ultimate results of such treatment are not very encouraging.—*London Medical Recorder*, March 20, 1890.

Intubation of the Larynx.—DR. G. HUNTER MACKENZIE describes the following cases illustrative of the complications which may arise during intubation of the larynx (*British Medical Journal*, May 24, 1890).

CASE I.—A child of twenty-one months intubated for membranous croup (laryngeal diphtheria?). The insertion of the tube was immediately followed by great relief, and all went well until six hours later, when profuse bleeding from the mouth and nose began, and the child very quickly died. Post-mortem examination showed a blood-clot firmly plugging the tube, and an erosion of the inferior laryngeal artery and vein. The erosion was evidently not caused by the tube.

CASE II.—A child of three and a half years suffering from "croup" (no mention of false membrane is made). The immediate results of the operation were excellent, the child breathing easily for seventy-two hours. At the end of this time he suddenly died, and on removal the tube was found blocked by a firm dry purulent mass.

CASE III.—In this case the insertion of the tube was difficult and, as Dr. Mackenzie doubted that the tube was in the larynx he did not remove the silk cord, but fixed it externally by means of adhesive plaster. A few hours before death the child gnawed through the cord, and at the *post-mortem* the tube was found in the lower part of the œsophagus. The difficulty in introduction was due to supra-glottic narrowing and distortion of the larynx.

Dr. Mackenzie has in a few instances seen the tube expelled by coughing, but writes that this need not cause alarm as there is usually sufficient time for reinsertion.

Since his experience with the second case above reported he has made it a rule to remove and examine the tube within forty-eight hours after its insertion. If dyspnœa then returns, which is the exception, the tube is replaced. The difficulty in swallowing is another reason why the tube should not be left in position longer than absolutely necessary.

Dr. William Hales in the same number of the *British Medical Journal* reports one hundred cases of intubation with 38 per cent. of recoveries and with seven consecutive successful cases.

Nitrite of Amyl in After-pains.—WINTERBURN employs nitrite of amyl for the relief of severe after-pains. His method of using it is as follows: A small piece of tissue-paper is saturated with five or six drops of the nitrite, then placed in a two-drachm vial, which is tightly corked.

The patient is directed to take out the cork and inhale from the bottle when the pains are severe.—*Archives of Gynecology*, June, 1890.

Powder for Fœtid Perspiration.—

℞.—Powdered rice . . . 2 ounces.
Bismuth subnitrate . . 7 drachms.
Potassium permanganate 3 "
Powdered talc . . . 1½ " —M.

To be dusted upon the perspiring parts.—*College and Clinical Record.*

Prescription for Removal of Comedones.—MCCASKEY uses the following application in the treatment of comedones:

℞.—Sulphuric ether . . . 8 drachms.
Ammonium carbonate . 1 drachm.
Boric acid . . . 20 grains.
Water, sufficient to make 2 ounces.—M.

To be applied locally twice daily.—*Peoria Medical Monthly.*

Ferruginous Tonic Pills.—The following formula is quoted in the *Archivesof Gynecology:*

℞.—Powdered digitalis, Ext. of nux vomica, Carbonate of iron } of each 8 grains.
Sulphate of quinine . . 1 drachm.
Ext. of gentian, sufficient quantity.

Mix and divide in thirty pills, of which one should be taken three times daily.

Prescription for Cases of Pin-worms.—The *Pittsburgh Medical Review* quotes the following formula, to be used in cases of pin-worms:

℞.—Tincture of rhubarb . . 30 minims.
Magnesium carbonate . 3 grains.
Tincture of ginger . . 1 minim.
Water . . . . 1½ drachm.—M.

Three or four such doses should be given in twenty-four hours.

THE MEDICAL NEWS.

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SATURDAY, JULY 5, 1890.

GONORRHŒA IN WOMEN.

THE serious complications which may result from gonorrhœa in women have only been fully recognized within very recent years. Noeggerath's statement that over one-half of the women of New York have some disease the result of gonorrhœa, seemed very much exaggerated when it was first made; but the results of abdominal surgery have strengthened this opinion.

Gonorrhœa in women is not only a cause of salpingitis, and resulting sterility and invalidism, but it is not infrequently the cause of death. The mortality of untreated cases of pyosalpinx is difficult to determine; we have seen it variously estimated at from ten to twenty-five per cent. In a consecutive series of fifty cases of pyosalpinx which were operated upon, we have found general peritonitis present in six cases. These six women were in a dying condition, and they alone, if not subjected to operation, would have given a mortality of twelve per cent. for this series of cases—a mortality certainly much too small when we consider the dangers to which the remaining forty-four women were constantly exposed. It is often difficult to estimate the part played by gonorrhœa in the production of salpingitis. The great majority of women who have pyosalpinx have been exposed to gonorrhœal contagion; but they have also been exposed to other causes of pyosalpinx, a very frequent one of which is criminal abortion. The presence of the gonococcus in the Fallopian tube is pathognomonic; but we believe that there are many cases of salpingitis caused primarily by gonorrhœa, in which careful search fails to reveal the specific diplococcus. This opinion is based on the examination of many specimens of tubal disease, with an undoubted clinical history of gonorrhœa, in which no gonococci were discoverable.

A case recently reported by DR. E. CAPPI (*Gaz. Hebd. des Sciences Méd. de Montpellier*, May 24, 1890) is of interest in this connection. A woman was operated upon for encysted peritonitis, and the gonococcus was found not only in the pus from the abdomen, but also in the discharge from the os uteri. Though the operation failed to show the primary cause of the abdominal abscess, yet it most probably originated from the rupture of a pyosalpinx of gonorrhœal origin. This case followed the usual course; the sequence of events being: vaginitis, endometritis, salpingitis, and closure of the Fallopian tubes; and finally, encysted peritonitis with the formation of a pelvic or an abdominal abscess, or general diffuse peritonitis following rupture of the tubal abscess. The case of peritonitis from gonorrhœa reported by DR. PENROSE in this number of THE MEDICAL NEWS followed a very unusual course. The progress of the disease was not delayed by local peritoneal adhesions resulting in closure of the Fallopian tubes. We know of no case with a similar history; and though the gonococcus was not discovered, yet the clinical history points so definitely to the cause that we must conclude that the disease was of gonorrhœal origin.

This case exemplifies in a forcible way the dangers of gonorrhœa in women. The physician should always remember that neglected gonorrhœa is dangerous not only on account of the consequences to the male, such as urethral stricture, cystitis, and epididymitis, but also on account of the much more frequent and more serious sequelæ which may occur in the female.

TRICHLORACETIC ACID IN INTRA-NASAL DISEASES.

THOUGH the galvano-cautery has to a great extent supplanted chemical agents in the treatment of hypertrophic rhinitis, in many cases the cicatrix produced by the application of pure chromic acid is sufficient to cause contraction of the turbinated

bodies, and possesses the advantages of not alarming the patient and of requiring comparatively little manipulative skill. Its disadvantages are that unless considerable care is exercised the acid spreads, causes an unnecesarily large slough and an undesirable amount of inflammatory reaction.

Trichloracetic acid, recently introduced in Germany as a cauterant, apparently has all the advantages and none of the disadvantages of chromic acid. VON STEIN and STANISLAW, of Moscow (*Journal of Laryngology and Rhinology*, June, 1890), recommend it not only in the treatment of hypertrophy but in that of acute nasal catarrh (coryza), ozæna, adenoid vegetations, and of various laryngeal diseases. Making due allowances for the enthusiasm of the experimentalists, their assertions are are still so positive that the drug should certainly be given a fair trial, although we believe its field will eventually be restricted to the cauterization of hypertrophies and possibly as a stimulant in the treatment of ozæna.

As a cauterant a crystal of the pure acid should be applied by means of a probe, precisely as chromic acid is ordinarily used, and, of course, during cocaine anæsthesia.

In ozæna a weak solution (five-tenths of one per cent.) should be rapidly brushed over the diseased mucous membrane. It is stated that this treatment, if repeated daily, corrects fœtor, softens the crusts and prevents their formation.

CORRESPONDENCE.

"DETACHMENT AND EXPULSION OF THE VAGINAL PORTION OF THE CERVIX DURING LABOR."

To the Editor of THE MEDICAL NEWS,

SIR: Under the above title, in the last issue of your journal, Dr. Hirst reports a case which is of considerable interest. The treatment of the case, however, strikes one as somewhat peculiar. Is it considered orthodox practice in Philadelphia to allow a multiparous woman to remain in labor attended by "vigorous uterine contractions," supplemented by "energetic abdominal action," for *twelve* hours, or, in fact, until the "vaginal portion of the cervix" is extruded from the vulva, without extending to her a helping hand? Could not the os uteri have been dilated artificially? And, if other means failed, could not the tissues surrounding the os (even though they had undergone "cicatricial infiltration") have been subjected to multiple incision (*débridement multiple*), and then the delivery have been carefully effected with forceps under chloroform? Dr. Hirst says: "the absence of serious symptoms after an injury of this gravity is remarkable." It is still more remarkable, in view of the practice adopted, that the body of the uterus did not rupture before the cervical portion gave way under the influence of the *vis a tergo*. Dr. Hirst seems to think that the recovery of the patient, without "serious symptoms," speaks well for the *asepsis* of the Maternity Hospital. We think that it speaks well for the *vis medicatrix naturæ*, and are inclined to attribute the recovery of the patient more to good luck than good management.

A READER OF THE NEWS.

June 16, 1890.

Before printing this note the Editor of THE MEDICAL NEWS submitted it to Dr. Hirst for reply, which is appended:

To the Editor of THE MEDICAL NEWS,

SIR: As I did not see the case reported by me in the last number of THE NEWS until after the cervix had been detached and expelled, the rather harsh criticism of "A Reader of THE NEWS" applies to the conduct of the resident physician in charge. Her action did not appear so heinous to me as to this critic. Would he, in a first stage of labor, which had only lasted twelve hours, uncomplicated by rise of the contraction-ring, by elevation of temperature, by marked disturbance of pulse, by anything, in fact, but delayed dilatation of a rigid cervical canal—would he, I ask, resort to the forced dilatation of the cervix, or to its incision? If so, he would find his "meddlesome midwifery" more dangerous than the conservative course pursued by the young graduate of not a year's standing, who was in charge of the case.

BARTON COOKE HIRST, M.D.

PTOMAINES AND BACTERIA.

To the Editor of THE MEDICAL NEWS,

SIR: My attention has just been called to a short article on ptomaines by Professor Ernst, of Harvard, in Volume V. of the *Annual of the Universal Medical Sciences*, 1889, and while the entire article might justify some comment, I am certainly at a loss to understand what authority Professor Ernst has for saying that Mr. Novy and myself in our little volume on *Ptomaines and Leucomaines* make "the suggestion that bacteria may be the *products* of these alkaloids." Will Professor Ernst be kind enough to inform me on what page of the volume I will find the suggestion? As the author of that portion of the volume in which a ptomaine is defined and its nature discussed, I had never heard of the "suggestion" until I had the pleasure of reading the article by Professor Ernst. Respectfully,

V. C. VAUGHAN, M.D.

The above having been submitted to Professor Ernst, he replied as follows:

To the Editor of THE MEDICAL NEWS,

SIR: The statement to which I referred and to which Dr. Vaughan wishes to be directed will be found in *Ptomaines and Leucomaines, or the Putrefactive and Physiological Alkaloids*, by Victor C. Vaughan, M.D., and Frederick G. Novy, page 86, sixth line from the bottom, reading "we are justified in saying that the microörganism may be an accompaniment or a consequence of the disease."

My attention was first called to the statement by a review of the book in the *American Journal of the Medical Sciences*, October, 1888, page 394.

Respectfully,
HAROLD C. ERNST, M.D.

TYPHOID FEVER IN CHATTANOOGA.

To the Editor of THE MEDICAL NEWS,

SIR: In an interesting paper by Dr. James E. Reeves, of Chattanooga, Tennessee, on "Some Points in the Natural History of Enteric or Typhoid Fever," read before the Association of American Physicians, in May last, and published in THE MEDICAL NEWS of June 7th, the author claims for Chattanooga a remarkable and almost entire immunity from typhoid fever, and quotes several well-known physicians of that city to prove that typhoid fever is a disease of extremely rare occurrence—one physician, in active practice for more than thirty years, having seen only three or four cases within that period.

The official records of the Chattanooga Board of Health, as favorable as they are, scarcely justify these extraordinary claims. The reports of the board charge 107 deaths to typhoid fever during the last four years. This mortality probably represents over 1000 cases of the disease. At all events, there appears a marked discrepancy between Dr. Reeves's conclusions and the official figures of the Health Department.

I observe, furthermore, that the Board of Health calculates the rate of mortality upon an estimated population of 40,000 instead of 60,000, the estimate adopted by the writer of the paper.

In studying the natural history of any disease as influenced by local conditions and environment, the official figures adopted by competent persons charged with the responsibility of collecting and publishing statistical data are entitled to serious consideration, and should outweigh, it seems to me, the random estimates of individuals, however capable and honest they may be.

The line between facts and mere opinions should be sharply drawn, and the opinions, to be trustworthy, should be supported by the facts; otherwise confusion may ensue, innocent persons may be misled, and evil may come when only good was intended.

Respectfully,
JAMES B. BAIRD, M.D.

ATLANTA, GEORGIA.

IODOFORM AND IODOL IN DIABETES MELLITUS.

To the Editor of THE MEDICAL NEWS,

SIR: I have just read a very interesting inaugural thesis by Dr. Fructuoso V. Valdés, presented last year to the National School of Medicine of Mexico. The subject of Dr. Valdés' essay is "Iodol," and in it he reports the excellent results obtained, by the local use of the drug, in the treatment of ulcers in general. The observations were made in the different hospitals of the Mexican capital, under the supervision of the ablest clinical professors. Among other cases, he reports, through the kindness of Dr. José Olvera, of the City of Mexico, in whose private practice they occurred, two of diabetes mellitus, in one of which the most flattering success followed the use of iodoform, in the other from the use of iodol.

I will translate the details of both cases, as they are extremely interesting, one of them corroborating the results obtained in a case recently reported by me.[1]

Dr. Olvera writes as follows: "Last year I attended a lady about fifty years of age, who was then suffering from glycosuria accompanied with polyuria (about 2½ litres of urine daily), general debility, intense thirst, and a very disagreeable dryness of the mouth and fauces. The first time that the urine was analyzed by Professor Morales it gave 33 grammes of glucose and 30 grammes of urea per thousand of the liquid. The patient, four years previously, had suffered for six months from nephritis with albuminuria. She recovered completely, but afterward had a rheumatic facial paralysis of the right side. It was impossible for me to ascertain, at the time of consultation, the length of time since she began to have the diabetic affection, nor the cause of the disease. She was submitted to a rigorous diet, excluding all sugary and amylaceous food, but was allowed to use bran bread. I prescribed a daily dose of 0.005 milligramme of strychnine, and at the end of the second month the 33 grammes of glucose determined by the first analysis had only been reduced by 2 grammes. I then administered a combination of iodoform and strychnine. The third time the urine was analyzed, the reduction was very noticeable, as the liquid gave only 18 grammes of glucose, which encouraged me to continue the treatment. At the end of three months the sugar had *entirely* disappeared from the urine. I deemed it prudent to continue the same diet and medicines for a month and a half longer, after which she was allowed to use gradually amylaceous and sugary articles of food. Notwithstanding the free ingestion of these in the course of time, no glucose could be detected by later analyses, a fact which greatly strengthens my belief that it was, undoubtedly, a case of perfect cure.

"Mrs. R. L. presenting an emphysematous condition and symptoms of a slow, progressive medullary sclerosis, began to notice last December an increase in the secretion of urine, especially at evening, which was the cause of sleepless nights. At first she attached no importance to this circumstance and attributed the disturbance to cold; but the continuous and gradually increasing dryness of the mouth and fauces decided her to consult me at the end of January last. The urine was analyzed by Professor Morales, who found 29 grammes of sugar per thousand of the liquid. The amount of urine passed in twenty-four hours was calculated to be 1200 grammes. Remembering the good results obtained from the combined use of iodoform and strychnine in the previous case, I immediately proceeded to treat the present one with the same agents. These were administered during one week only and then, according to the suggestion of Dr. Valdés, I decided to substitute the iodoform with *iodol*, but continuing to give strychnine. The analysis of March 3d gave only *three grammes* of glucose per thousand of urine. The total amount of urine passed in the twenty-four hours has also diminished considerably. It is, perhaps, needless for me to say that the patient's diet has been properly regulated.

"From what has been here stated I think we ought to

[1] THE MEDICAL NEWS, vol. lvi., March 8, 1890.

congratulate ourselves on the happy results obtained in the latter case. In the first case it may be that, while the diminution of the glucose was slower under the use of iodoform and strychnine than that of the second case under iodol and strychnine, the more rapid disappearance of the sugar was due not to the action of the drugs, but to the constitution of the patient, the nature of the cause or causes of the disease, idiosyncrasies and what not; however, setting these considerations aside, we have sufficient reason to continue our experimentation in this matter, and further experience will, in the future, decide the usefulness or worthlessness of the drug in question."

I can only add that there is, undoubtedly, sufficient clinical evidence to show that iodol deserves further and continuous trials at the hands of the profession, and not simply as a substitute for iodoform but as a drug possessing its own and highly valuable therapeutical properties.

I am about to make an experimental physiological study of iodol, which will form the substance for a future article. DAVID CERNA, M.D.

Laboratory of Experimental Therapeutics,
University of Pennsylvania.

NEWS ITEMS.

A New Hospital at Zanzibar.—The Emperor of Germany has subscribed 20,000 marks to help in the establishment of a new German hospital at the capital of Zanzibar, in East Africa.

University of Berlin.—*The Lancet* announces that the chair of mental pathology at the University of Berlin, made vacant by the death of Professor Westphal, has been accepted by Dr. Grashey, of Munich.

Protection of the Insane against Fire.—In view of the frightful loss of life from the burning of asylums at Montreal, Canada, and Utica, N. Y., the Committee of Charities of the Massachusetts Assembly have framed a bill requiring iron fire-escapes to be constructed on the outside of all asylums for the insane throughout the State. Suitable apparatus for the distribution of water-hose within the buildings will be required, and the trustees are directed to cause a regular monthly inspection to be made of these life-saving appliances.

The Death-rate of England.—The death-rate in England in 1889 was 17.9 per 1000; in 1888, 17.8 per 1000. For each of the nine years, 1881–1889, the rate was lower than in any year prior to 1881, and the average annual rate for that period was only 18.9 per 1000. For the ten years, 1871–1880, the average annual rate was 21.4 per 1000. This shows a saving of 2.5 in every 1000 of the population, comparing the last two decades with each other. The Registrar General of England estimates that not less than 600,000 people in England and Wales at present survive by reason of the declining mortality rate—that is, if the rate 21.4 per 1000 had persisted in the past nine years instead of falling to 18.9, there would have been 600,000 more deaths.

This improvement is no doubt largely referable to the improved sanitary condition of the United Kingdom, more especially in the great cities. One proof of this is seen in the decreasing mortality from zymotic diseases, such as smallpox, scarlet fever, and typhoid fever. Infant mortality, also, has shown a marked decline, and is another index of the life-saving results of an improved sanitation.

OFFICIAL LIST OF CHANGES IN THE STATIONS AND DUTIES OF OFFICERS SERVING IN THE MEDICAL DEPARTMENT, U. S. ARMY, FROM JUNE 24 TO JUNE 30, 1890.

By direction of the Secretary of War, leave of absence for two months, to take effect August 6, 1890, is granted to WILLIAM N. SUTER, *First Lieutenant and Assistant Surgeon.*—Par. 3, *S. O. 149, A. G. O.*, June 26, 1890.

By direction of the Secretary of War, leave of absence for three months and fifteen days, to take effect as soon as his services can be spared, is granted WILLIAM C. BORDEN, *Captain and Assistant Surgeon*—Par. 11, *S. O. 46, A. G.O.*, June 23, 1890.

Captain HOWARD CULBERTSON (Retired) died Juue 18, 1890, at Zanesville, Ohio.

Appointments in the Medical Department, U. S. Army, to be Assistant Surgeons, with the rank of First Lieutenant, June 6, 1890:

KEEFER, FRANK R., of Pennsylvania, vice Woodruff, promoted.

RAYMOND, THOMAS U., of Indiana, vice Newton, resigned.

SNYDER, HENRY D., of Pennsylvania, vice Wilson, resigned.

SMITH, ALLEN M., of New York, vice Matthews, promoted.

HEYL, ASHTON B., of Pennsylvania, vice Hall, promoted.

CLARKE, JOSEPH T., of New York, vice Porter, resigned.

By direction of the Secretary of War, CHARLES R. GREENLEAF, *Major and Surgeon*, will attend the encampment of the Pennsylvania National Guard at Mount Gretna, Pennsylvania, from the 18th to the 26th of July, 1890, for the purpose of accompanying the Surgeon General of Pennsylvania in his inspection of the camp.—Par. 11, *S. O. 144, A. G. O.*, June 20, 1890.

OFFICIAL LIST OF CHANGES IN THE STATIONS AND DUTIES OF THE MEDICAL CORPS OF THE U. S. NAVY, FOR THE TWO WEEKS ENDING JUNE 28, 1890.

PAGE, JOHN E., of Berryville, Va.—Commissioned Assistant Surgeon in the Navy.

KENNEDY, ROBERT M., of Pottsville, Pa.—Commissioned Assistant Surgeon in the Navy.

WHITFIELD, JAMES M., of Richmond, Va.—Commissioned Assistant Surgeon in the Navy.

STONE, LEWIS H., of Litchfield, Conn.—Commissioned Assistant Surgeon in the Navy.

ATLEE, LOUIS W., *Assistant Surgeon.*—Detached from the U. S. S. "Marion, and granted three months' leave.

OFFICIAL LIST OF CHANGES OF STATIONS AND DUTIES OF MEDICAL OFFICERS OF THE U. S. MARINE-HOSPITAL SERVICE, FROM MAY 31 TO JUNE 21, 1890.

GASSAWAY, J. M., *Surgeon.*—When relieved at Cairo, Ill., to proceed to New Orleans, La., and assume command of the Service at that station, June 4, 1890.

STONER, G. W., *Surgeon.*—Granted leave of absence for three days, June 18, 1890.

WASDIN, EUGENE, *Passed Assistant Surgeon.*—Granted leave of absence for fourteen days, June 5 and 10, 1890.

WHITE, J. H., *Passed Assistant Surgeon.*—To proceed to Savannah, Ga., on special duty, June 9, 1890.

HEATH, F. C., *Assistant Surgeon.*—Granted leave of absence for fifty-eight days, June 10, 1890.

MAGRUDER, G. M., *Assistant Surgeon.*—Granted leave of absence for twenty days, June 2, 1890. Ordered to examination for promotion, June 5, 1890.

WOODWARD, R. M., *Assistant Surgeon*—Relieved from duty at Chicago, Ill., to assume command of Service at Cairo, Ill., June 4, 1890.

CONDICT, A. W., *Assistant Surgeon.*—Upon expiration of leave of absence, to report to Medical Officer in command at Chicago, Ill., for duty, June 4, 1890.

RESIGNATION.

HEATH, F. C., *Assistant Surgeon.*—Resignation, to take effect August 31, 1890, accepted by the President, June 10, 1890.